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RESULTS OF TOTAL THYROIDECTOMY IN HEART DISEASE*

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SUFFICIENT time has now elapsed to evaluate the benefit of the total thyroidectomies performed on 27 patients with heart disease in this clinic. We were rather slow in adopting this procedure, first, because it seemed to us to be unsound physiologically, and secondly, because we had had some experience with cases of angina pectoris with myxedema in which we could not alleviate the annoying symptoms of one disorder without aggravating the symptoms of the other. However, because of the encouraging reports concerning patients in whom this procedure had been carried out, and after actual interviews with such patients, the first operation for total thyroidectomy was performed on a patient with heart disease in this clinic in December, 1933. Since that time 27 patients have been operated upon, the last operation taking place 1 year ago.

It seems unnecessary to discuss here the theories proposed by the various advocates of this form of treatment. The results are, after all, the important aspect of this question and the purpose of this paper is to report on them in a strictly critical manner.

SELECTION OF PATIENTS

Patients who had had adequate medical care but who were still incapacitated to a considerable degree were selected for the operation. While all of the patients with congestive heart failure were not bedridden, they were unable to carry on their ordinary labors, and all had signs of congestive failure with the exception of case 1. In the angina pectoris group those patients selected for operation had definite and severe angina pectoris. All were not incapacitated, but they were unable to work without difficulty. While there were two main groups, those with congestive heart failure and those with angina pectoris, some cases fell into both groups.

OPERATION

The operative procedure was well tolerated in most instances. There was 1 postoperative death that occurred in a patient with mitral stenosis and severe congestive heart failure (case 25). This patient was failing progressively following her first break in cardiac compensation. Despite the usual medical treatment and, finally, venesection, she failed to improve, and the operation was done in an unwise attempt to stop the patient's downhill course. Death occurred 15 hours afterward. A postmortem examination was not performed, but death was apparently due to congestive heart failure and pulmonary thrombosis. Unilateral vocal cord paralysis occurred in 2 cases as a complication of the operative procedure. No tetany occurred after the operation in any case.

POSTOPERATIVE MYXEDEMA

In 19 of the cases one-half grain of thyroid was given immediately after the operation in an attempt to ward off some of the distressing symptoms of myxedema. Because of this, it was impossible to judge the severity of myxedema that might otherwise have developed. Varying degrees of this condition were seen although they were not always comparable to the level of metabolism. A sense of stiffness and generalized aching was troublesome to some of the patients. Hypersensitivity to cold, although frequently present, was never a distressing symptom. A determination of the blood cholesterol was made before and after the operation and was found to follow the variation in basal metabolic rate quite evenly, i.e., the blood cholesterol increased as the myxedema developed.

CIRCULATION RATE AND HEART SIZE

The circulation rate of the patients' blood was determined before and after the operation by the decholin method. The findings were not at all consistent, but there was usually an increase in the velocity of blood flow in the patients with congestive heart failure when compensation was restored. A slow velocity of blood flow caused by congestive heart failure was usually found before the operation. In the patients with angina pectoris the velocity of blood flow was changed from a normal circulation time to a prolonged circulation time owing to the development of myxedema. In the patients with congestive heart failure who showed no improvement after the operation there was either no change in the already slow circulation rate, or it was made even slower.

A 7 foot heart plate was taken on the majority of the patients before and after the oper-

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ation, and no significant change in heart size was noted, except in one case (case 13). This patient had angina pectoris with a history of congestive heart failure. He had only mild congestive failure before the operation, at which time his heart measured transversely 14.3 cm. Four months after the operation the heart measured transversely 12.5 cm. The transverse diameter of the chest was the same as before the operation. In this patient the vital capacity increased slightly from 2.3 to 2.8 liters. Mild myxedema had developed, although he was taking one-half grain of desiccated thyroid.

RESULTS IN ANGINA PECTORIS

Total ablation of the thyroid was done in 14 patients with angina pectoris, in 2 of whom there was associated congestive heart failure. Eight of the patients had a background of hypertension, 4 had rheumatic heart disease with valvular lesions (one of these had associated hypertension), and 2 patients had neither hypertension nor rheumatic heart disease. In this group there were no operative deaths. Since the operation 3 patients have died (cases 10, 14 and 20). The first 2 patients had very similar histories. Both had rheumatic heart disease with an aortic lesion and both died suddenly 4 months and 3 months, respectively, after the operation. In these 2 patients there was no improvement before death. In the third there was moderate improvement for 1 year and then complete recurrence. The 11 living patients have been followed from 1 to 2½ years. Improvement has been slight in one case, between 25 and 50 per cent in 5 cases, and over 50 per cent in the remaining 5 cases. Case 7 has been classified as showing no improvement although there was some definite improvement in that the patient could do a considerable amount of clerical work for 1½ years after the operation. At present, however, he is in the same condition as he was before the operation, but feels that it helped him because during the past 2 years his pain has been milder than it had been previously. The remaining 9 patients have shown a distinct improvement that has varied from merely a change of the type of pain, to almost complete relief from the angina. The degree of improvement in these patients appears to have diminished slowly, since the first year after the operation.

RESULTS IN CONGESTIVE HEART FAILURE

Total ablation of the thyroid was done in 13 cases of congestive heart failure. The underlying factor was rheumatic heart disease with valvular lesions in 8 cases, syphilitic heart disease in 2, and hypertensive heart disease in 3 cases. There was one operative death in this group, as mentioned above. Since the operation 6 other patients of this group have died and the causes of their deaths are given in the case histories. An evaluation of the results of the operation in this group is more difficult to make than in the group of patients with angina pectoris because of the unpredictable course of congestive heart failure. There was no real improvement in 8 of these cases. There was definite, although temporary, improvement in one case and there was definite and gratifying improvement in 4 cases (15, 16, 19 and 24). Armour's desiccated thyroid was used to control symptoms of myxedema in all of the patients.

TABULATION OF RESULTS

Table 1 lists the cases, the length of time since operation and a few remarks about each case. Results are classed A, B, C, D, as suggested by Clark, Means and Sprague:

A. No recurrence of signs or symptoms. Activity increased. Excellent result.
B. Symptoms less severe with increased activity. Moderately improved.
C. Symptoms less severe without increased activity. Slightly improved.
D. No improvement. Poor result.

CASE HISTORIES

A brief summary of each case is given below and the result is noted at the end of each summary.

1. **H. C., aged 45 years, female. This patient did not have persistent congestive heart failure but had had recurrent attacks of paroxysmal dyspnea and intermittent mild congestive heart failure associated with mitral stenosis and auricular fibrillation. Control of her symptoms with digitalis was uncertain because of her sensitivity to the drug. Her circulation rate was 30 seconds (*arm mouth*) before the operation. It is obvious, therefore, that she had some heart failure at this time. She felt completely cured after the operation and resumed normal activity until she died suddenly 14 months afterward. The cause of death was not determined, but we felt that it was probably the result of pulmonary embolism. (Grade A until death.)**

2. **B. G., aged 53 years, female. This patient was admitted to the hospital with severe congestive heart failure, marked edema and free fluid in the abdomen. The congestive failure had been present for 2 months, and there was no improvement from a regime of digitalis, diuretics and rest. The patient had angina pectoris of 7 years' duration, which had become progressively worse. The heart trouble was based on hypertensive heart disease with coronary sclerosis. The blood pressure on entry was 210 systolic, 110 diastolic. When no improvement followed the operation, one grain of Armour's thyroid was given to the patient, as well as digitalis and diuretics as used previously. There was very little improvement, and the patient was discharged. She was admitted 1 year later with severe congestive heart failure and with myxedema. On small doses (1 gr.) of thyroid and diuretics (salyrgan) she improved slowly but, in the**

TABLE 1

	Age	Sex	Angina	Congestive Failure	Length of Time Followed	Results A	B	C	D	Dead	Remarks
1.	45	F		1	14 mos.	1				? Embolism	Very mild case of failure. Excellent results.
2.	53	F		1	11 mos.				1	Ruptured diverticulum	No real benefit—severe case.
3.	39	M	1		2¼ yrs.				1		Slightly improved at first.
4.	44	F		1	2½ yrs.				1		Severe case—tapping before and since operation.
5.	40	F		1	2 yrs.				1		Severe case—no improvement; tapping every 2 wks. In bed.
6.	61	F		1	14 mos.				1	Heart failure	Severe case, no aid before death.
7.	48	M	1		2 yrs.		1?		1?		For 1½ yrs. patient able to do clerical work, which he could not do before, later return of symptoms.
8.	51	F	1		2 yrs.		1				Does housework whereas formerly was incapacitated.
9.	47	F	1		2 yrs.			1			Does housework; was incapacitated before.
10.	39	M	1		4 mos.				1	Acute heart failure	Atypical angina with aortic stenosis—heart weighed 800 gm.
11.	60	M	1		2 yrs.		1	1?			Can do much more—mild attack with marked exertion.
12.	47	F	1		2 yrs.			1			Good result—moderately severe angina.
13.	60	M		1	2 yrs.		1				Had severe angina—appreciative of pain relief, which is partial.
14.	41	M	1		3 mos.				1	Acute heart failure	Same as case 10.
15.	43	F	1		2 yrs.		1	1?			Definite and good improvement. Was a mild chronic failure.
16.	25	M		1	4 mos.		1			Acute heart failure	Severe failure—improved until sudden death.
17.	49	M	1		21 mos.			1			Fair result, atypical angina.
18.	56	F	1		1¼ yrs.		1				Good result—working.
19.	57	M		1	18 mos.			1			Severe case—slight improvement.
20.	56	F	1		21 mos.				1	Congestive heart failure	Less frequent and severe attacks for 1 year. Then symptoms returned.
21.	48	M	1		2 yrs.			1			Good result at first, poor now.
22.	59	F		1	6 mos.				1	Carcinoma, pancreas and liver	No help, severe case and had carcinoma. Died 6 mos. postoperative carcinoma.
23.	35	F		1	1½ yrs.				1		No help—severe case.
24.	21	M		1	1 yr.	1?	1?				Pulmonary hemorrhages and marked dyspnea—recovered. Working.
25.	34	F		1	Postop.				1	Operative death	Severe case—going downhill rapidly.
26.	50	M	1		1 yr.			1			Moderately severe angina improved, more active, less pain.
27.	46	F		1	3 mos.				1	Congestive heart failure	Syphilitic aortic regurgitation. Severe failure. Should not have been done. No improvement.

course of improvement, developed an "acute abdomen" and died. Postmortem examination showed a ruptured diverticulum of the colon. The heart weighed 640 gm. Advanced coronary sclerosis was present. (Grade D—died.)

3. A. L., aged 39 years, male. This patient's main complaint was angina pectoris and, in this case, it was associated with rheumatic heart disease and mitral stenosis. Although he had some dyspnea, his ability to work was greatly limited by pain. Definite signs of myxedema developed postoperatively, and 6 months after the total thyroidectomy he was given ½ gr. of thyroid daily. This controlled the distressing symptoms of myxedema. At first the patient was improved, he could do more work with less pain, but at the present time, 27 months after the operation, he is no better. He has both angina pectoris and congestive heart failure. (Grade D.)

4. J. T., aged 44 years, female. This patient had had rheumatic fever five times and was known to have had rheumatic heart disease for 17 years. One year before entry she had congestive failure, and an abdominal paracentesis was performed for the first time, after which her course was gradually downhill. She had mitral stenosis, aortic regurgitation and congestive heart failure on entry. It was felt that this patient was developing abdominal fluid more rapidly than could be accounted for by the heart failure, and the additional cause seemed to be cirrhosis of the liver from chronic passive congestion. After total thyroidectomy there was temporary improvement, as was shown by a lengthening of the time between necessary abdominal paracenteses, but at the present time, 2½ years later, the patient is incapacitated by congestive heart failure. Thyroid extract has been used to control the symptoms of myxedema, but the patient has distressing gastric symptoms which may be partly the result of thyroid deficiency. (Grade D.)

5. E. T., aged 40 years, female. Dyspnea and palpitation had been present in this patient for 5 years, and evidence of congestive heart failure came on about 5 months before entry. She had rheumatic heart disease with aortic regurgitation and was unable to do her work owing to congestive heart failure because of which a total thyroidectomy was performed. After the operation there was slight, immediate relief, but the patient then developed more congestion than before operation, and abdominal paracentesis became necessary. Since that time she has been in a constant state of congestive heart failure. She has had 62 abdominal taps since the operation 2 years ago. A complete failure. (Grade D.)

6. M. W., aged 61 years, female. This patient was admitted to the hospital with congestive heart failure of 2 months' duration. She had syphilitic aortitis with aortic regurgitation and cardiac enlargement. Very little, if any, improvement was obtained from the use of diuretics, bed rest and antisyphilitic treatment for a period of 4 weeks. In an attempt to stop the downhill course a total thyroidectomy was performed. A complication of the operation was injury to the right recurrent laryngeal nerve with vocal cord paralysis. The downhill course of the patient was not altered perceptibly and she died 14 months later of congestive heart failure. (Grade D—dead.)

7. F. P., aged 48 years, male. This patient was moderately obese, with a history of severe attacks of angina pectoris, for 1½ years, with two attacks suggestive of coronary occlusion in the previous six months. It was impossible to decide definitely if he had had previous coronary occlusion. He was incapacitated by anginal pain on both exertion and rest, so that he was unable to do his work, which was clerical. His heart was moderately enlarged, and his blood pressure 150 systolic, 100 diastolic. After a total thyroidectomy was performed, the patient continued to have anginal attacks, although of much less intensity. He was able to do one half of his usual work until a month or so ago, and now 2 years after the operation, he has pain that is as severe and incapacitating as that he had had previously (grade D) now; (grade B)—temporarily.

8. H. F., aged 51 years, female. For 5 years this patient had moderately severe angina pectoris. The symptoms had become worse in the previous 4 to 6 months so that she could do very little housework and nothing outside the home. She had used nitroglycerin and caffein derivatives for relief. Hypertension was present as was also mitral stenosis. After a total thyroidectomy, the patient was given ½ gr. of thyroid daily. Improvement in this case was definite and it was gratifying to the patient. At the present time, 2 years later, she does her housework and also a small amount of outside activity. She has very rare mild anginal attacks and does not require nitroglycerin to control them. (Grade B.)

9. R. W., aged 47 years, female. This patient had severe angina pectoris of one year's duration. She was incapacitated and was limited to a chair and bed existence. There was no congestive heart failure. Her blood pressure was 195 systolic, 140 diastolic. It was felt that the basis of her disorder was hypertensive heart disease with coronary sclerosis. After a total thyroidectomy, the patient was given ½ grain of thyroid daily. For 8 months she felt a remarkable improvement (75 per cent) until she developed what seemed to be an acute coronary occlusion (not observed by us). After recovery from this episode she was not so well and she has had a return of anginal pains of a moderately severe degree. She is not confined to the house and does some of her own housework. She feels 25 per cent improved, 2 years after operation. (Grade C.)

10. L. C., aged 39 years, male. This patient complained of unbearable anginal pain of 6 months' duration, and stated that morphin was used to relieve him at times. There was no congestive heart failure. Aortic stenosis and aortic regurgitation were found on examination and the heart was greatly enlarged. One-half grain of thyroid daily was given to the patient after a total thyroidectomy. After his discharge from the hospital the patient did more work and said he was free from pain, but, through his family, it was learned that he still had the same pain. Three months after the operation, the patient drank a good deal of whiskey and came into the hospital complaining of extreme anginal

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pain. He was obviously drunk. It was necessary to use morphin and amytal to relieve his pain. He developed pulmonary congestion and died in 2 days. At postmortem examination, the heart was found to be enlarged (800 gm.). A hard, scarred aortic valve with stenosis was found. The coronary arteries were entirely normal throughout. (Grade D—died.)

11. W. W., aged 60 years, male. For 2 years this patient had had angina pectoris, but was not incapacitated. He was able to work a few hours daily, but mild exertion would bring on attacks of pain. Hypertension was present; the blood pressure being 210 systolic, 110 diastolic. After a total thyroidectomy was performed the patient had no real pain but some feeling of constriction that came on with exertion. He was given ½ gr. of thyroid daily and is still taking it. At present, 2 years later, he feels that he is still greatly improved by the operation, although he is not working. (Grade B—? C.)

12. M. L., aged 47 years, female. This patient had had dyspnea and attacks of angina pectoris on exertion for 4 or 5 years. These symptoms had been definite although not very severe. She had been able to do part of her housework. Coronary heart disease was the basis of attacks of the angina pectoris. The heart was not enlarged. The blood pressure was 154 systolic, 80 diastolic. After a total thyroidectomy was performed ½ grain of thyroid was given daily. She noted considerable improvement in the attacks of angina. She could do her own housework and exert herself more in climbing stairs, without pain. The good result of her operation continues. She is 50 to 75 per cent improved, 2 years postoperatively. (Grade B.)

13. H. P., aged 60 years, male. For 2 or 3 years this patient had had severe, but not incapacitating attacks of angina pectoris. One year before entry he had had congestive heart failure, from which he recovered after a few weeks in bed. Since that time he had had angina so severely that he had been incapacitated and completely bedridden. The basis of his heart trouble was hypertensive and coronary heart disease. The blood pressure was 160 systolic, 105 diastolic. One-half grain of thyroid was given after a total thyroidectomy. This patient received complete relief from his severe angina, but had an occasional minor attack of substernal oppression. Fifteen months after the operation he returned to the ministry (his occupation) but the work proved to be too strenuous, and he required 4 weeks of rest in bed to recover. At present, 2 years after the operation, he is still free of severe attacks of pain, but he is not able to work or to be very active. (Grade C.)

14. H. C., aged 41 years, male. This case resembles case 10. The patient had rheumatic heart disease with aortic stenosis, aortic regurgitation and severe attacks of angina pectoris. The angina was of one year's duration and was not improved after a month of rest in bed. The patient was unable to pull weeds in the garden for 10 minutes without pain. During the previous 2 months he had had occasional bloody sputum and dyspnea on exertion. Recovery from the total thyroidectomy was slow, and there was a persistent cough. This patient did not do well at home. He was incapacitated, with cough and dyspnea although the pain was less severe. He died suddenly 3 months after the operation. The cause of death is not known to us, but, from a description, it probably was either pulmonary embolism or acute congestive heart failure. (Grade D—died.)

15. C. N., aged 43 years, female. For 4 years this patient had had symptoms and signs of congestive heart failure. She had not been incapacitated, but had had a great deal of difficulty doing her housework. She had edema and pulmonary congestion on entry, which had been constantly present over a year prior to her admission. She had refused to go to bed on account of her household responsibilities. The heart condition was caused by rheumatic heart disease, mitral stenosis, aortic regurgitation and auricular fibrillation. The results of a total thyroidectomy in this case were gratifying. The patient was kept on ½ grain of thyroid daily to control the symptoms of myxedema. She went home, free of congestion, and did a great deal more work than she had been able to do before the operation. She has remained free of congestive heart failure and, at the present time, 2 years later, is doing her housework regularly. (Grade B.)

16. R. W., aged 25 years, male. Eighteen months prior to entry, this patient had had congestive heart failure due to rheumatic heart disease with mitral stenosis and auricular fibrillation. He had been in bed 10 weeks after this first break in compensation, from which time until the operation, he always had had some evidence of heart failure, although he had not stayed in bed and had been able to do a small amount of work. He developed congestion of the liver and lungs. Before the operation he was treated with rest in bed for 3 weeks, with no relief of incapacitating congestive heart failure. After a total thyroidectomy, he had a good diuresis and became greatly improved. In a month he was able to go upstairs slowly and to sleep comfortably at night. He had no congestive heart failure but 4 months after the operation he died suddenly of what was probably acute heart failure. The operation probably was not justified in this case, but there was considerable improvement before his death. (Grade B—dead.)

17. F. S., aged 49 years, male. This patient had had angina pectoris for one and one-half years. It had been relieved by nitroglycerin even up to time of operation, but the attacks had been so frequent and the threshold of activity so low, that the patient was incapacitated. The basis of his trouble seemed to be coronary heart disease. The patient was given ½ grain of thyroid daily after a total thyroidectomy. He had less frequent and less severe attacks after the operation and was able to do some work. The ultimate relief after 21 months, however, was not more than 25 per cent. (Grade C.)

18. G. L., aged 56 years, female. For one year prior to entry this patient had had definite but only moderately severe attacks of angina pectoris. She had not been incapacitated, having been able to do some of her housework. She had had a discrete adenoma of the thyroid, which was not toxic. The basal metabolic

rate was minus 6. It was felt desirable to remove the discrete adenoma in any case and total thyroidectomy was therefore advised in an attempt to alleviate the anginal attacks as well. The patient had hypertension, and her blood pressure was 230 systolic, 115 diastolic. After operation the patient could do more work with greater freedom from pain, and the result, in this respect, has remained the same 15 months postoperatively. (Grade B.)

19. I. R., aged 57 years, male. This patient had been completely incapacitated as a result of arteriosclerotic and hypertensive heart disease with congestive failure. He had had 4 or 5 periods of decompensation in the past 4 years despite good medical care. His blood pressure was 160 systolic, 110 diastolic. After a total thyroidectomy, the patient was given ½ grain of thyroid daily. At first he said that he was 100 per cent improved, but clinically he seemed only about 25 per cent better, and he has remained about the same ever since. At present, 18 months postoperatively, he can walk around but does not work. He requires digitalis and diuretics. (Grade C.)

20. G. T., aged 56 years, female. This patient had had severe typical angina pectoris of two years' duration. She had had hypertension of unknown duration. The blood pressure was 180 systolic, 110 diastolic. She had been incapacitated with the angina, having had as many as six attacks a day, and sometimes even at night when in bed. She had had good medical treatment, but nitroglycerin and other drugs had brought only temporary relief. This patient received definite, although only moderate, relief from a total thyroidectomy for one and one-half years. She then began to have as much difficulty as she had had previously and died 21 months after the operation. The cause of death was acute congestive heart failure. (Grade D.)

21. M. H., aged 48 years, male. This patient was a farmer and had been used to hard manual labor. He had had angina pectoris for 4 years. At first the attacks were mild, and nitroglycerin gave relief, but in the previous year he had been incapacitated for work because the attacks became so severe. Hypertension of a moderate degree was present. The blood pressure was 190 systolic, 100 diastolic. The results of a total thyroidectomy in this case were good at first. The patient was able to do about one-half of his usual work and still remain free from angina. He was given ½ grain of thyroid daily to control myxedema. At the present time, however, 2 years later, he can do no work and he has pain on exertion to almost the same extent as he did before the operation. (Grade C.)

22. F. S., aged 59 years, female. This patient had had dyspnea as well as a chronic type of edema for 5 or 6 years. In the past year she had been incapacitated. In addition to the above difficulty, she had had pain in the abdomen, which seemed to be from congestion of the liver. The basis of the cardiac trouble was hypertension and arteriosclerosis. She had auricular fibrillation. The patient did not improve after a total thyroidectomy. She died 6 months after the operation, and postmortem examination showed a carcinoma of the pancreas with metastases of the liver. The heart was enlarged and coronary sclerosis was present. (Grade D—died.)

23. M. D., aged 35 years, female. This patient had a history of congestive heart failure of 1½ years' duration, and ascites of sufficient degree to require 15 abdominal taps during the previous year. Salyrgan and other diuretics had been used. The underlying heart lesion was mitral stenosis and aortic regurgitation with auricular fibrillation. After a total thyroidectomy the intervals between the necessary abdominal paracenteses seemed longer, temporarily, but later the fluid collected as rapidly as before. At the present time, 1½ years postoperatively, she is tapped every 15 days. (Grade D.)

24. H. D., aged 21 years, male. For 4 years this patient had had moderate dyspnea, but had been able to do his work. During the 15 months before his operation, the patient had had 3 severe attacks of intrapulmonary hemorrhage, which seemed based on the cardiac lesion of mitral stenosis. After each hemorrhage the patient had had further attacks of dyspnea. On his last entry, prior to the operation, he had had extreme dyspnea and incipient congestive heart failure. Since the total thyroidectomy the patient has been able to do his work and seems stronger. He has less dyspnea, and for 12 months has had no recurrence of pulmonary hemorrhage. He complains of stiffness and pains in the legs, which seem due to myxedema that is partially controlled with ½ grain of thyroid daily. (Grade A or B.)

25. A. D., aged 34 years, female. This patient was the only operative death. She was admitted with severe congestive heart failure from mitral stenosis. There was no improvement with rest in bed, digitalis, salyrgan, and venesection. A total thyroidectomy was done as her condition seemed at a standstill. The operative course was uneventful and she was returned to her room in about the same condition as she was before operation. Her blood pressure dropped, however, and she died in 15 hours, probably of congestive heart failure, although she may have had pulmonary vein thrombosis or embolism. (Grade D—died.)

26. T. C., aged 50 years, male. This patient had had attacks of angina on exertion to such an extent that he had not been able to do his work. Also, he had had attacks after walking short distances, and after eating. His blood pressure was normal, and the heart was not enlarged. A total thyroidectomy was done in an attempt to get him in condition for work. After operation he noted considerable improvement. He could exert himself without pain, but he developed distressing symptoms from myxedema, and an attempt to control this caused a recurrence of the angina. He is at present (one year later) taking ½ grain of thyroid daily and is improved to such an extent that he is usually free from pain, although he cannot work. (Grade C.)

27. M. F., aged 46 years, female. Early signs of congestive heart failure had been manifest in this patient for one and one-half years before entry. Two months before entry she began to have obvious congestive heart failure with edema. Her cardiac difficulty was syphilitic heart disease with aortic regurgitation. (Blood pressure 200 systolic, 80 diastolic.) Ordinarily this patient would not have been operated upon, but there was some uncertainty relative to possible hyperthy-

roidism. She had had a consistently elevated basal metabolic rate, the average being plus 60. For this reason the operation was performed. The thyroid showed adenomatous changes but no hyperplasia. The patient did not improve clinically, and her basal metabolic rate showed no significant change. She died of congestive heart failure 3 months after the operation. (Grade D.)

DISCUSSION

In chronic conditions, such as congestive heart failure and angina pectoris, patients are apt to grasp at any type of treatment even though it may be of doubtful value. The favorable results obtained are frequently subjective. Because of the hope instilled by the possibility of help, the physician must display sound judgment in the selection of patients for such a procedure as total thyroidectomy. The attitude of the patient and his family is usually as follows: "You may do anything that offers the slightest chance of relief." This is said without a consideration of the financial burdens to be added to the already large potential debt of chronic illness. Thus, it hardly seems justifiable to advise the procedure unless a definite relief of pain or a return to more activity can be expected.

Our results after operation have been fairly satisfactory in some cases. This appears to have been owing in large part to the fact that many of the patients that were selected for operation were not extremely ill. In some cases they might have been symptom-free while on a regime of partial rest and inactivity. It is important to remember that the average patient is a great deal more careful in following instructions in regard to rest and work after an operation than before. The patient feels that since he has incurred the expense of such a procedure, he must obey every instruction to make it successful. He often does not feel this way about the usual medical regime advised or urged before any operative procedure is carried out.

In following the patients with angina pectoris after a total thyroidectomy, it has been obvious that the amelioration of pain is a more or less temporary affair. We have had to change the grading of the results on several occasions because of favorable effects at first and less satisfactory results later. This does not necessarily discredit a therapeutic procedure, because the progression of the disease process must be considered in tabulating results over a period of years. It would be impossible to designate a certain period of time for amelioration of symptoms as justifying an operation, and another period as being not justifiable. When attacks of angina pectoris are held in abeyance by a total thyroidectomy for only 6 months in a patient that will live several years, the operation would seem to be unjustified. If a patient on whom total thyroidectomy had been done, remained free of pain until death 6 months later, however, the procedure might be considered worth while, provided that myxedema, itself, did not contribute to this death.

The decrease in the number of total thyroidectomies performed at present is indicative of a swing back from an enthusiastic trial of a new procedure. Physicians caring for patients at home after the total thyroidectomy, are noting, at times, that these patients are in a condition similar to that present before the operation, and consequently they are not so anxious to have other patients undergo such a procedure. This judgment of the family physicians who are intimately associated with the patients must weigh heavily in the evaluation of this treatment. It seems almost impossible to keep the patient from having more hope than is obviously justified after a total thyroidectomy. Although the plain facts are stated and the possibility of improvement is not magnified, he builds up a great hope, and it is distressing to see the reaction in those that get no relief or very little relief from the operation. Such patients now feel that there is no further aid for them and that they are doomed. This has not been a uniform attitude, but it has been noted enough times to make it worthy of mention.

Our conclusions differ but little from those of Clark, Means, Sprague and others. There are some patients on whom the operation of total thyroidectomy seems justifiable; however, they are but few in number.

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THE AGE OF HARVARD MEDICAL SCHOOL IN RELATION TO THAT OF OTHER EXISTING MEDICAL SCHOOLS IN THE UNITED STATES

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SINCE the observation of the tercentenary of Harvard College, which is without question the oldest college of arts in the United States, has recently occurred, it seems timely to inquire about the relative ages of the older existing medical schools.

The statement is commonly made that four medical schools were established in the United States in the eighteenth century, namely, those of the University of Pennsylvania, King's (Columbia) College, Harvard College and Dartmouth College.

This statement is less than one half complete since five other medical schools were established before the beginning of the nineteenth century. These were the Medical School of the College of Philadelphia, established in 1765 and finally closed in 1791; the Medical Department of the College of William and Mary, established in 1779 and continued for three or four years; the Medical Department of the University of the State of Pennsylvania, established in 1783 and closed in 1791; the Medical Department of Queen's College, established in 1792 and closed in 1828; and the Medical Department of Transylvania University, established in 1799 and closed in 1857.

Four of these nine schools may be dismissed with relatively brief mention, for three do not exist and have no definite successors and the fourth is clearly younger than the Harvard Medical School.

THE MEDICAL SCHOOL OF THE COLLEGE OF WILLIAM AND MARY

When Thomas Jefferson became governor of Virginia in June, 1779, he planned to make the College of William and Mary of Virginia into a state university. Under his influence as one of the "visitors" of that college, it was reorganized into a series of *schools*, each being the equivalent of what is now designated as a department of a college of arts. For example, there was a School of Mathematics in this reorganization. Two new subjects and schools were added at this time—a School of Law, the first in the American colonies or the United States, and a School of Anatomy, Medicine and Chemistry.[1, 24, 25] Each of these schools had a single professor. Dr. James McClurg (1747-1823), a native of Virginia, who had been a student in arts at the College of William and Mary and who had received his degree in medicine at Edinburgh in 1770, was appointed professor of anatomy and medicine in 1779 and held the professorship until 1783. His annual salary was "eight hogsheads of tobacco". Of the exact number of his students, there is no record.[3, 21]

When the field of action of the Revolutionary War encompassed Williamsburg, the college suspended teaching. When teaching was resumed after the war, instruction in anatomy and medicine was omitted since Dr. McClurg had moved to Richmond. This institution granted but a single medical degree, which was an honorary degree. In 1782 the degree of Doctor of Medicine was conferred on John F. Coste, first physician of the French Army, which was operating in that region.[24]

This was the third medical school to begin instruction, and the first to expire. It may be said to have been an infant victim of the War, and fittingly, it might be given the epitaph once a favorite on the tombstones of infants:

"If I was so soon to be done for
Oh, what was I born for?"

THE MEDICAL DEPARTMENT OF QUEEN'S COLLEGE

Nicholas Romayne was born in New York City in 1756 and was a student of arts in King's College when the Revolutionary War began. He went to Europe and received his M.D. degree at Edinburgh in 1780. After two years of study in Paris, he returned to America, practiced for two years in Philadelphia and returned to New York City in 1784.[21]

In 1784 King's College was reorganized under the name of Columbia College and Nicholas Romayne, not yet thirty years of age, became its youngest trustee. An attempt coincidentally to revive the medical school failed. A year later, in 1785, Dr. Romayne and three others were made professors of medical subjects, but instruction did not get under way.

Having found his two offices of trusteeship and professorship to be inconsistent, Dr. Romayne resigned his professorship in 1787, and because he was dissatisfied with this inactivity of Columbia College regarding its medical school, established a private school for teaching medicine, which attracted many students from a wide area.

In January, 1791, he and six other young physicians applied to the Regents for a charter for an independent medical school. This application was refused on the ground that the Regents had no power to grant a charter for a medical school. In March, 1791, the legislature specifically gave them such power, but because of opposition from professors and other officers

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of Columbia College and from some of the older physicians, the Regents, in February, 1792, finally refused to grant a charter to Dr. Romayne and his associates.[9, 14]

In April, 1792, Dr. Romayne applied to Queen's College in New Brunswick, New Jersey, asking that he and his associates be made a medical faculty to conduct instruction in New York City. With three others he was appointed to conduct examinations and to recommend candidates for medical degrees. These men were not actually appointed as professors, although later general catalogues of this college carry their names as such.[9, 26] Upon the recommendation of these men, Queen's College conferred seven medical degrees in 1792, and eight in 1793.[26] The appointments of these examiners or professors terminated in 1794. This ended the first phase of the Medical Department of Queen's College.

Nicholas Romayne, at that time the first President of the Medical Society of New York County, was the leader in securing a charter and in organizing the College of Physicians and Surgeons in New York City in 1807. He became its first president and a professor. In 1811, however, controversies arose that resulted in his resignation, both from his presidency and his professorship. At the same time four other professors resigned. This was the first instance in American medical education of a procedure that became commonplace in later years—the secession of part of a medical faculty to form a rival institution.

In 1812 Queen's College appointed Dr. Romayne and six others to professorships and constituted them a medical faculty to conduct a medical school in New York City, where a total of twenty-one medical degrees were conferred from 1812 to 1816, inclusive.[26] Coincident with the serious illness of Dr. Romayne in 1816, which resulted in his death in 1817, the second phase of this medical school ended in September, 1816, when instruction was suspended, although the professorial appointments were not formally annulled until 1826.[26]

In 1820 another period of dissension began in the College of Physicians and Surgeons in New York City. This resulted in the resignation of the entire faculty of six in April, 1826.[8] The seceders endeavored to have Columbia College revive its medical school under their guidance, but failed.[15] In October, 1826, four of these men and two others were appointed as the Medical Faculty of Rutgers College,[11] the name of Queen's having been changed to Rutgers in 1825.[9] Instruction began in November, 1826, with one hundred and forty-two students enrolled, while the College of Physicians and Surgeons had but eighty-six.[15] The opponents of this new school went to the New York Legislature, and in April, 1827, that body passed the following act: "Be it enacted by the People of the State of New York, represented in Senate and Assembly, That all diplomas granted by authorities out of the state to individuals who have pursued their studies in any medical school in this state, not recognized by the laws of the state, shall be void and of no effect, as it regards authorising the said individuals to practice physic and surgery in this state."[9, 15, 30]

The Rutgers Medical Faculty then sought protection under an institution that was chartered in New York, and secured affiliation with Geneva College. From 1827 to 1830 they gave instruction under the name of the Rutgers Medical Faculty of Geneva College. The catalogue of this institution for 1828-1829 shows one hundred and thirty-six students.[27] The last session of the affiliated faculties was from 1829 to 1830, although the professorships under Rutgers College were terminated in 1828. Rutgers College conferred thirty-six medical degrees in 1827, and a few honorary degrees until 1835. In the three phases of medical education under Queen's and Rutgers a total of seventy-nine medical degrees were conferred, of which twenty-two were honorary.[9, 26]

The Geneva Medical Institution, which was organized in 1834 and continued until 1872, was not a continuation, either in time or in personnel, of the affiliation of Geneva College with medical teaching in New York City in 1827. Furthermore, there was no connection between the Rutgers Medical Faculty and any other medical school. It can be said, therefore, that the Medical Department of Queen's or Rutgers College, had no successor.

THE MEDICAL DEPARTMENT OF DARTMOUTH COLLEGE

The medical school of Dartmouth College, the second medical school in New England, was founded in 1797 and began instruction in 1798.[5] Its first medical degrees were awarded in 1798. It is now in its one hundred and thirty-ninth year. Since 1914 it has not given instruction in the clinical courses of the curriculum, and so does not give a degree in medicine. It is clearly younger than the Harvard Medical School and need not be further considered in this article.

THE MEDICAL DEPARTMENT OF TRANSYLVANIA UNIVERSITY

In 1780 the legislature of Virginia set aside land to establish a seminary of learning in its colony, the District of Kentucky. Elementary teaching began in 1785, and by 1789 there was a grammar school with thirteen pupils. In 1796 an academy was established, and in December, 1798, a charter united the grammar school with the academy under the name of Transylvania University, located at Lexington, Kentucky. In January, 1799, the trustees of

this institution established the Medical Department of Transylvania College and appointed two professors, both of whom accepted the appointment and began to teach in December, 1799. It is of interest to know that these men received salaries. One of them continued to teach until 1806. A third professor was appointed in 1801, and another in 1805.[20] There is no reliable record of the number of students in these early years. There were no medical graduates before 1818.[20]

Instruction in this school was somewhat irregular for ten years and was practically suspended for another seven years. In 1817 there began a thorough reorganization,[20] which opened an era of marked success. From 1824 to 1835 this school had the second largest enrollment in the United States, having reached a peak of two hundred and sixty.[2] It was closed permanently in 1857, when the number of its graduates was 1,881.[20] It was the last of the medical schools to be organized in the eighteenth century and was the last to expire. It had no definite legal successor, although there is a shadowy succession through its personnel to the schools in Louisville. For a time the same group of teachers conducted a medical school at Louisville in one season of the year and at Lexington at another season, an example of the favorite method, which was in vogue in several regions, by means of which a student was able to attend the required "two courses of lectures" and be graduated within a single calendar year.

We now come to the main subject of this paper, which is a consideration of the relative ages of the oldest existing medical schools in Boston, New York and Philadelphia. In order to present all the factors in the problem, it will be necessary to follow in some detail the sequence of events that relate to the early medical schools in each of these three localities.

Inasmuch as the significant distinction between a college or university and other educational institutions is the chartered right to grant degrees, it appears that the date of issue of a charter that confers the power to award degrees is the best single criterion of the date of birth of a college or university.

In the case of the professional schools, which were later established under the broad charters of colleges or universities, however, the date of the charter of the parent institution is not logically the date of birth of the professional schools. The best criterion for the date of birth of a professional school appears to be the date when instruction to its students was begun, such instruction having been given by a man appointed by trustees to a teaching position, the designation of which included a subject that was distinctly of professional name and nature, and only after that man had accepted such appointment. These limitations aim to exclude as an acceptable date for the beginning of a medical school the date of prior private teaching by men who later became members of a faculty in which some or all of the members taught professional subjects.

THREE EIGHTEENTH CENTURY MEDICAL SCHOOLS IN PHILADELPHIA

The charter of the College, Academy, and Charitable School of Philadelphia bears the date of 1755.[7, 17, 23] The Academy had been chartered six years earlier. The first degrees were granted in 1757. Among the founders and the earnest supporters of this institution was Benjamin Franklin, who was its first president.[23] This institution is usually called the College of Philadelphia rather than by its longer, legal name.

John Morgan and William Shippen were both born in 1736 in Philadelphia and later attended the same preparatory school. Morgan was graduated from the College of Philadelphia in its first class in 1757,[4, 21, 28] Shippen, from the College of New Jersey (Princeton), then at Newark, in 1754.[4, 21] They were students at the same time in Edinburgh, where both took their medical degrees. While there, they agreed to found a medical school in Philadelphia.[4, 21]

Shippen returned in May, 1762, and in the autumn of that year began private medical teaching to a class of twelve students. Morgan returned in 1765 and proposed to the trustees of his alma mater that he and Shippen be appointed professors to constitute a medical faculty. In this he was supported by a letter from the head of the Penn family in England.[4, 21] This was done, and instruction in medicine began in the autumn of 1765. This marks the date of the first institutional medical instruction in the United States under men whose professorial titles included definitely medical subjects. The first degrees were conferred in 1768. There was interruption in instruction, which was partial in some years, total, in others, owing to the Revolutionary War. "The medical course fell into disuse after 1773 or 1774."[8]

The Province of Pennsylvania became the Commonwealth of Pennsylvania by adoption of a constitution on September 28, 1776. The sessions of the General Assembly of 1777 and 1778 were occupied with matters of emergency among which was an act, passed in March, 1778, which seized the estates of "traitors", that is, loyalists or tories.[7, 17] A year and a half later, on November 27, 1779, an act that chartered the University of the State of Pennsylvania was passed.[7, 17] It is to be noted that the name, University of Pennsylvania, was not considered to be sufficient, but pride in the new statehood extended to the insertion of the word, "state", in the name of the university.

To support the new university, the Pennsylvania Assembly dedicated "such and so many

of the confiscated estates, yet unsold and unappropriated as shall appear necessary" not to exceed a yearly income of "fifteen hundred pounds, computing wheat at the rate of ten shillings per bushel.[7, 17]" Not satisfied with having endowed the university by the method of confiscation, they extended their activities and confiscated the College Academy, and Charitable School of Philadelphia. Its charter was abrogated, and all its "estates, real, personal, and mixed, claims and demands, . . . all evidences, mortgages, specialties, deeds and instruments . . . and the library and philosophical apparatus," were turned over to the new university.[7, 17]

The preamble of this act stated that the reason for the abrogation of the charter and seizure of the property was that the trustees of the College of Philadelphia had, in 1764, passed a by-law that "narrowed the plan of free and unlimited catholicism of the founders".[7, 17] The by-law had specified that the Provost and Vice-Provost should not be of the same church affiliation, and it was strange that its dire threat was not discovered until fifteen years after its passage. This purported reason was, of course, only a subterfuge. The real reason was that some of the trustees of the College of Philadelphia were suspected of having some sympathy with the crown and with the local loyalists.[4, 6, 23] It was essentially a political contest between the conservatives and radicals of that day.

The University of the State of Pennsylvania attempted to organize a medical school by electing to professorships all the members of the former medical faculty of the College of Philadelphia. With the exception of one man, they all refused the appointments.[4] Repeated attempts were made to organize a medical faculty by a series of appointments, all of which were refused. Although outlawed as a faculty, some, if not all, of the former medical professors continued to give private medical instruction, and they maintained a pro forma faculty organization until the events of 1789.

After four years the University of the State of Pennsylvania succeeded in assembling a medical faculty, which began instruction in November, 1783.[4, 6] This date marks the beginning of the Medical Department of the University of the State of Pennsylvania.

Although it had no medical faculty and was giving no regular or complete medical course, the University of the State of Pennsylvania conferred a total of nineteen medical degrees in 1780, 1781, 1782 and 1783.[4, 28] These degrees were awarded after examination by the "former medical professors", who were not at this time professors of the University. The examinations followed instruction that had been given mainly under private auspices. These degrees were for the most part those of Bachelor of Medicine, usually the initial degree in course. Perhaps these degrees "by examination" should be considered as more in the nature of honorary degrees than of degrees in course.

When the act that confiscated the College of Philadelphia was passed by the assembly in 1779, Benjamin Franklin was in Europe as Ambassador to France. Upon his return in 1785, he took up the cause of the institution in whose founding he had taken so large a part,[4, 23] and owing largely to his prestige and efforts, the assembly on March 6, 1789, repealed the parts of the act of November 27, 1779, "as touch or in any wise concern or relate to said antient corporation", that is, the College of Philadelphia. The trustees were "reinstated and restored" with possession of all endowments, property and equipment. The reason given for the repeal was that the original seizure was "without trial by jury, legal process, or proof of misuser or forfeiture" and was "repugnant to justice, a violation of the constitution and dangerous in its precedent to all incorporated bodies".[7] The University of the State of Pennsylvania was left with little more than its annual income to the value of three thousand bushels of wheat from the estates of the traitors.

The declaration by the Assembly of the Commonwealth that the seizure of the College of Philadelphia was unconstitutional shows conclusively that the University of the State of Pennsylvania was not the legal successor of the College of Philadelphia, and that the medical department of that university had no valid claim to succession to the medical department of the college by the legislative act of 1779.

The act of March, 1789, made two rival medical schools in Philadelphia. In view of what had passed, there was anything but cordial relations between them. This continued for two years, the medical department of the College of Philadelphia having the larger attendance.

This antagonistic situation was unfortunate for both institutions, and on September 30, 1791, the Assembly of the Commonwealth passed an act that chartered a new institution under the name of the University of Pennsylvania.[19] The language of the act shows that it is not an amendment to either of the two former charters, but a new charter for a new institution; the personnel, trustees, and faculties were chosen equally from those who held similar positions in the two former institutions, and all the assets of both the former institutions went to the new one.

In the conferences between the representatives of the two institutions, which took place preliminary to the agreement that resulted in the new charter, there was discussion as to the name to be adopted. The University of the State of Pennsylvania wished to retain that name. This would mean that it had absorbed the College of Philadelphia. The College of Philadelphia wanted the name to be University of Philadelphia, tak-

ing one word from the name of each institution. Finally, the name, University of Pennsylvania, was agreed upon as more likely to secure state aid. This discussion is evidence that the new institution was not the continuation of either of the older institutions, but a new one formed from both.

This legislative act terminated both the College of Philadelphia and the University of the State of Pennsylvania, and established the University of Pennsylvania. Hence, the present Medical Department of the University of Pennsylvania, under that name and under the present charter, dates from 1791. However, the medical faculty was not appointed until January, 1792. The first medical degrees of the University of Pennsylvania were conferred in 1792. Information is not available as to the date when instruction began; but under the limitations of the criteria of beginning of a medical school, which were set down in a previous paragraph, the Medical Department of the University of Pennsylvania dates from the early months of 1792. It was, therefore, older by a few months than the medical department of Queen's College.

There is succession of medical instruction in Philadelphia, with interruptions partial or complete, which began in 1772 and ended in 1783; but in the eighteenth century in that city there were three different medical schools under three different charters and names—the College of Philadelphia (1765-1791), the University of the State of Pennsylvania (1783-1791) and the University of Pennsylvania (1792-).

EARLY MEDICAL SCHOOLS IN NEW YORK CITY

In anticipation of a charter issued on October 31, 1754 to establish King's College, instruction was begun in July 1754.[18] The first degrees were conferred in 1758.[29] In 1763, action toward establishing a medical school was begun, but not completed. In 1765 Dr. Samuel Clossy became a teacher in natural philosophy at the college[29] and, in the same year, he gave some lectures in anatomy, although his teaching title did not include this subject. In 1767, a medical faculty of six was appointed, and teaching was begun in November of that year, with three students. The first medical degree was conferred in 1769. Hosack says, "a regularly constituted medical school was not completed until 1769".[31] No medical degrees were conferred from 1774 to 1793 as medical instruction was much interrupted after 1774, when the faculty had become reduced by death and resignation.

In 1776 the Revolutionary War brought suspension of all activities of the college. It is difficult to ascertain just how much medical teaching was done from 1774 to 1776, but it was irregular and incomplete. In 1784 the educational system of the state was organized under the name of the University of the State of New York, a nonteaching organization that consisted of regents, and King's College was revived under the name of Columbia College.[16] A medical faculty was appointed but did not function.

In 1791 coincident with the rebuilding of the New York Hospital which had been burned while being erected just before the war, a new attempt was made to revive the medical school and new faculty appointments were made.[14] Instruction began in the early weeks of 1792. There were five graduates in 1793 and in twelve of the following seventeen years there were from one to four graduates per year, the last being in 1810. The total number of graduates of the Medical Department of Columbia College was thirty-four. The attendance varied, the highest being sixty-three in 1791-1792 and the lowest fifteen in 1799-1800.[29] After the session of 1809-1810 the instruction was irregular and incomplete. In 1813 the school was permanently closed, and the professorships terminated by vote of the trustees of Columbia College. This ended the medical department of Columbia College and for forty-seven years thereafter Columbia College had nothing whatever to do with medical education.

In March, 1807, under an act that had been passed by the legislature in 1791, the Regents granted a charter for a medical school to the Medical Society of the County of New York. In May, 1807, under this charter was established the College of Physicians and Surgeons in the City of New York, with one hundred and twenty members of the medical societies of the county and state as trustees. Instruction began in November, 1807.[8] This school was not the successor of that of Columbia College, but its rival.

Nicholas Romayne was president of the College of Physicians and Surgeons from 1807 to 1811. In 1811 there was a reorganization, and the hundred and twenty trustees resigned. Eleven of them and two other men were appointed to make the new board. At this time Dr. Romayne and three others resigned from the faculty.

In June, 1812, the College of Physicians and Surgeons of the Western District of New York was chartered at Fairfield, New York, nearly 200 miles from New York City.[16] This made four medical schools in the state, three of which were in New York City, and trebled the competition with the feeble medical department of Columbia College.

Instruction in the medical school of Columbia College was irregular from the end of the session of 1810-1811. The College of Physicians and Surgeons, in seeking to fill the vacancies on its faculty, which were occasioned by the resignations of 1811, invited some of those on the faculty of the medical department of Columbia College. The trustees of the latter college stated that they had no objection to the acceptance of this invitation by some of their medical professors, and as a result, some Columbia men

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joined the faculty of the College of Physicians and Surgeons at this time, although they still remained nominally on the inactive faculty of the older medical school.

During the next two years the trustees of Columbia College attempted to revive the medical school. In September, 1813, a committee of trustees reported to the Board that "a union had taken place between the said College of Physicians and the Professors appointed by this Board in the Medical Institution established under its authority", and, "that it will be of public benefit that the Board do approve of the Union aforesaid and do rescind the Resolution appointing a Medical Faculty in Columbia College." The resolution was rescinded.[22]

Some writers[11] have called this action a "union" or "consolidation" of the schools. If the first phrase of this report is interpreted as saying "the professors recently appointed have not accepted our appointments, but have joined our rival" we shall get nearer the actual facts. The trustees of Columbia College then withdrew the invitation to these men to become members of its faculty and abandoned the field of medical education. This minute of the trustees refers to a rival institution and to professors as individuals. There is no statement that the two medical schools were consolidated, and the inference is hardly warranted that by this minute the medical department of Columbia College can claim continuity through the College of Physicians and Surgeons during the period from 1813 to 1860.

John C. Dalton, writing in 1888, referred to the College of Physicians and Surgeons as "now in the eighty-second year of its existence". At that time he was president of the College of Physicians and Surgeons, and apparently did not consider that the events of 1813 made it possible to trace its origin to that of the medical department of Columbia College in 1767. He dates the establishment of the College of Physicians and Surgeons as 1807, and speaks of "combination of the faculties" in 1811, the medical lectures at Columbia having been discontinued". He does not, however, designate this combination of the faculties as a consolidation of the schools. Beck,[2] in referring to this period, says that the medical school of Columbia College was "discontinued", and makes no reference to union of the two schools.

In 1860 the College of Physicians and Surgeons formed an "alliance" with Columbia College. The terms of this arrangement gave the authorities of the latter no control over the choice of medical professors or medical teaching, but they agreed to approve certain phases of the actions of the separate corporation of the College of Physicians and Surgeons, and that college was permitted to add to its name the phrase, "Medical Department of Columbia College."

This loose affiliation continued until 1878 when endeavors to bring the College of Physicians and Surgeons under the control of the trustees of Columbia College began. It was not until 1891, however, that the medical college was actually transferred to the ownership and control of Columbia University.

Since there is an interval of forty-seven years, from 1813 to 1860, in the two phases of medical education under the charter of Columbia University, it seems somewhat gratuitous to interpret that the now existing medical school of that University began in the eighteenth century.

THE MEDICAL SCHOOL IN CAMBRIDGE AND BOSTON

"As early as 1764 evidences are found showing that the College Authorities at Harvard had in contemplation the establishment of a medical school in connection with that institution." These evidences are in connection with statements made when the library was burned in January, 1764.[10] That such a plan was acceptable must have been known to Dr. Ezekiel Hersey, a graduate of Harvard College, when he drew his will by which at his death in 1770 £1,000 became immediately available, and ten years later an additional £1,000, to endow professorships in subjects distinctly medical. This was the first endowment specifically created for medical teaching in the United States. No appointment of medical professors under the Hersey endowment was immediately made. The receipt of the additional £1,000 that was to come at the death of Dr. Hersey's widow was possibly being awaited. Moreover, any new development of medical education was overshadowed by the events of the Revolutionary War.

In May, 1782, the Corporation of Harvard College took the first effective step toward filling a medical professorship by appointing a committee, which submitted a report in September, 1782, outlining the establishment of a school.[10] This report was accepted, and on November 22, 1782, the corporation voted to establish three professorships and elected John Warren to one of these. In December Benjamin Waterhouse was elected to another,[10] and in May, 1783, Aaron Dexter was elected to the third.[10]

On October 7, 1783, Warren and Waterhouse were inducted into their professorships, and on October 16, Dexter. The induction lectures of these men may be considered as the beginning of instruction in the Harvard Medical School. It was one month previous to the beginning of medical instruction in the University of the State of Pennsylvania. Therefore, Harvard Medical School was the fourth medical school in the United States to be founded, having been preceded by those of the College of Philadelphia, Columbia College, and the College of William

and Mary; but since it is the only one of those four now in existence under the same name and charter, it becomes the oldest of the existing medical schools.

To recapitulate, the University of the State of Pennsylvania was not the legal successor of the College of Philadelphia by the acts of 1779, since in 1789 as much of this act as concerned the College of Philadelphia was declared by legislative act unconstitutional and, therefore, could not have been at any time legal. The University of Pennsylvania, which was chartered in 1791, was not identical with either of the older institutions and, obviously, could not be identical with both. The action of 1791 is subject to two interpretations as to the succession of the University of Pennsylvania to the University of the State of Pennsylvania and to the College of Philadelphia, for both of which interpretations arguments can be advanced. It is certain, however, that the medical department of the College of Philadelphia, under that name and by authority of the charter under which it was established in 1765, went out of existence in 1791, as did also the medical department of the University of the State of Pennsylvania, which had been established in 1783.

The events of 1813 regarding the College of Physicians and Surgeons and the medical department of Columbia College are also subject to two interpretations as to succession, but it is certain that, under its former name and by the authority of the charter of Columbia College, the medical department of Columbia College was discontinued in 1813. Since there was an interval of forty-seven years when Columbia College had no contact with medical education, the more logical interpretation appears to be that the present medical school of Columbia University is not the same as that which existed from 1767 to 1813.

Whichever of the interpretations just noted may seem preferable to any individual, it is nevertheless certain that the Harvard Medical School is the oldest existing medical school in the United States that has operated continuously under the same charter and with the same name.

SUMMARY

The medical schools that were established in the United States in the eighteenth century were nine in number as listed below in the order of their establishment. Those which no longer exist are indicated by an asterisk, and those in which instruction and operation were interrupted for periods during their existence are indicated by a dagger. The date at which instruction began precedes each name and the date of discontinuance follows. All were established under charters that were primarily for a college of arts, although sufficiently broad to include professional schools.

November	1765*†	College of Philadelphia	1791
November	1767*†	Columbia College	1813
?	1779*	College of William and Mary	1783
October	1783	Harvard College	Still in existence
November	1783*	University of the State of Pennsylvania	1791
January	1792	University of Pennsylvania	Still in existence
October	1792*†	Queen's (later Rutgers) College	1828
November	1798	Dartmouth College	Still in existence
December	1799*†	Transylvania	1857

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If anyone is moved to question the interpretation that Harvard Medical School was founded in 1783 instead of in 1782 as is usually stated, I call to witness John Warren and Aaron Dexter, professors in that school from its beginning. Their opinion of the date of founding is far more worthy of attention than the opinion of the present writer, but it happens that since the foregoing was written I have found that their opinions and mine coincide, unless the words *founded* and *established* are taken as having distinctly different meanings, as some may interpret them.

The records of Harvard College show that, in February, 1810, Doctors Warren and Dexter presented to the corporation a memorial regarding the moving of the medical school from Cambridge to Boston. A contemporary manuscript copy of that memorial, signed with the initials J. W. and A. D., is in the Harvard archives. Its opening phrase is, "The medical institution established in this University in 1783 . . ."

FRACTURES OF THE MANDIBLE AND MAXILLA*

BY JOSEPH A. DOHERTY, D.M.D.†

FRACTURES of the jaws are among the more commonly seen bone injuries. The increase in the number of these fractures since the World War is the result of more rapid means of transportation, the use of machinery, and greater participation in sports. The maxilla is much less frequently the site of fracture than the more exposed and vulnerable mandible. Because of the presence of the teeth, great accuracy in the reduction of these fractures is necessary in order to prevent malocclusion and preserve normal mastication. Good cosmetic and functional results follow only when certain basic principles are applied in the management of these cases.

SYMPTOMS

The symptoms of fracture are tenderness at the site of injury, swelling, ecchymosis, abnormal mobility and the loss of function. The most reliable diagnostic sign in fracture of the jaw is the inability to close the jaws in a natural manner. Clinical findings should always be confirmed by the x-ray. A complete roentgenographic survey should be made in all cases. Displacement of the fragments in a maxillary fracture is owing to the direction of the force and gravity. Though displacement in fracture of the mandible is primarily caused by trauma it is maintained or modified by the muscles to which the mandible furnishes attachment.[1] In a general way, unless the direction of the fracture line prevents displacement, the anterior fragment is depressed by the mylohyoid, geniohyoid, genioglossus and digastric muscles; while the posterior fragment tends to be elevated by the masseter, temporal and internal pterygoid muscles. Hemorrhage is, as a rule, easily controlled. Trauma to the infraorbital and inferior alveolar nerves often produces an anesthesia of the tissues that they innervate.

LOCATION OF FRACTURE

Fractures of the maxilla are usually caused by blows of a crushing nature, such as those which characterize automobile injuries. The injury may involve only a part of the alveolar process, or may be more extensive and accompanied by fracture of the malar and nasal bones. Transverse fractures can occur above the apices of the teeth through the maxillary antra or higher up when they involve the orbits. In such cases, the face tends to sag downward, and the bony framework is supported only by the soft tissues.

The most frequent site of fracture of the mandible is at or near the angle; the next most common location is in the region of the mental foramen. Bilateral fractures most often include both of these areas. Trauma to the chin may cause fracture of the symphysis and, not too infrequently, involvement of one or both condyles. Condyloid fractures with displacement cause a deviation of the median line to the affected side, and a shortening of the ramus

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on the same side naturally follows. Fracture of the coronoid process is not so rare as is sometimes supposed. Most fractures of the body of the mandible should be considered compound, since they usually communicate with the mouth.

TREATMENT

Treatment consists in the care of the soft tissues, and the reduction and maintenance of the fragments of the bone in proper position until union occurs. In selecting the method of im-

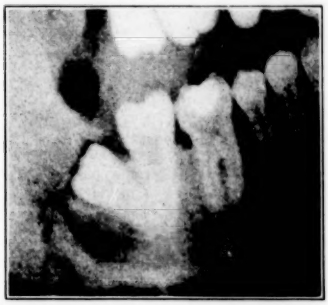

FIG. 1. Fracture of the angle of the mandible with elevation of the ramus and depression of the body.

mobilization, the following factors are important: the location of the injury, the amount of displacement, and the presence or absence of teeth. Since fixation is principally a mechanical problem of a dental nature, surgical intervention for bone wiring or plating is obviously quite limited in its application. The latter treatment is now regarded as obsolete, and direct wiring of the fractured parts is advisable

FIG. 2. Malocclusion of the teeth, characteristic of fracture of the mandible.

only in those cases of edentulous jaws where extensive displacement exists and other methods would not be successful.

The Barton bandage cannot be considered of value except as a temporary aid, or as a supplement to some other form of fixation. In a simple fracture of the mandible without disturbance of the normal occlusion, the application of a head bandage will usually be followed by a good result. Such cases, however, would do equally well without any stabilizing force. In

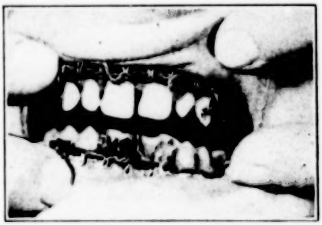

FIG. 3. Immobilization of a maxillary fracture previous to applying interdental wiring.

a fracture of the jaw with displacement, the Barton, or any other type of head bandage, not only fails to hold the fractured bones in position, but many times actually increases the dis-

FIG. 4. Reduction and fixation of a fracture of an edentulous mandible by means of the circumferential method.

placement.[2] Moreover, the pressure exerted by this bandage causes swelling of the soft tissues and discomfort to the patient.

The majority of cases of fracture of the mandi-

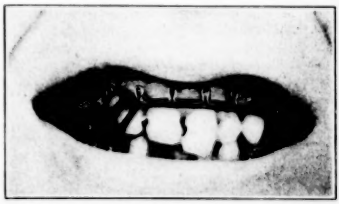

FIG. 5. Fracture of the symphysis reduced by the application of intraoral elastic force.

ble and maxilla can be successfully treated by nonsurgical intraoral methods. These consist in the use of splints, wires and elastics, which, when properly applied, will restore and main-

tain the normal occlusion of the teeth—a fundamental principle in the handling of this type of bone injury. Gilmer[3] was the first to suggest wiring the upper teeth to the lower teeth in treating fractures of the jaw. He devised two methods of immobilization, which form the basis for the various modifications that are employed at the present time. These are known as the Gilmer No. 1 and No. 2 methods. The former can be used most successfully where all or most of the teeth are present in both jaws; while the latter is applicable in those cases in which only a few teeth remain. Should it later be necessary to open the mouth, as in the treatment of an alveolar abscess or a complicating osteomyelitis, the second method is advantageously employed since no material interference with the main fixation splints results. During recent years there has been more frequent use of elastic traction as a means of reducing fractures of both the mandible and maxilla.[4] The ease with which badly displaced jaws or those in which treatment is delayed may be brought into correct alignment, when elastic force is properly applied, is most astonishing. Unless injuries of a more serious nature are present or the patient is in shock, early reduction and immobilization are advisable. In those cases in which treatment is delayed, the inflammatory reaction is more pronounced, and the development of a submaxillary or cervical phlegmon is more probable. The removal of infected teeth or roots in the line of fracture is an accepted procedure, provided that unnecessary trauma is avoided. If the fracture occurs at the angle, and the extraction of a tooth in the short fragment would cause the ramus to strike against the maxilla, it is better to allow the tooth to remain until fibrous union is sufficient to hold the parts in correct anatomic position.

In treating fractures of edentulous jaws, splints or artificial dentures may be employed to effect immobilization. The supplementary, extraoral, elastic force must be strong enough to prevent movement of the fractured bone. A fracture of an edentulous mandible with marked displacement may be treated by direct wiring or by the circumferential method. In the former method, an orthopedic or dental drill is used to perforate the bone well outside the fracture line, and a soft silver wire is inserted through the holes and tightened in crisscross fashion. When circumferential wiring is employed, skin incisions are first made at the inferior border of the mandible. An antrum trocar and cannula are then passed into the oral cavity, penetrating the soft tissues on both sides of the bone. A small wire is passed through the cannula in a manner so as to surround the jaw and a specially constructed vulcanite or metal splint. Twisting the ends of the wire reduces the fractured parts to normal position. The skin incisions are sutured, and as a rule, no noticeable scars remain after healing has taken place.

A consideration of injuries of the jaws would not be complete without mentioning fracture of the neck of the condyle. When displacement exists, there is a tendency for the head of the process to be drawn inward and forward, with resultant deviation of the chin toward the side of injury. Advocates of open operation have endeavored to restore the condyle to its former position in the glenoid cavity, but, owing to the strong pull of the external pterygoid muscle, this procedure is difficult. Moreover, injury to the facial nerve is not always unavoidable. Because of the fear of ankylosis, these fractures have also been treated by permitting mobility of the joint. When it is considered that malalignment of the teeth is most pronounced in these cases and that restoration of normal occlusion is the cardinal principle of treatment, the necessity for retaining the jaws in a fixed position during the healing process is most evident. In the past, good functional results have been obtained by re-establishing the former relation of the upper and lower jaws without manipulating the fractured condyle.[5] This can be brought about with such ease by means of interdental wiring that it seems not only hazardous, but unnecessary, to resort to open reduction and treatment by mobility.

The type of anesthesia to be employed in the treatment of fractures of the jaw depends on the physical condition of the patient, the amount of displacement and whether edema and infection are present. Local anesthesia is effective in the reduction of simple fractures, but the use of a general anesthetic will be found more satisfactory in complicated cases. Objections to ether have been based on the possibility of vomitus being aspirated during the postoperative period. If the application of the intermaxillary fixation wires is postponed until consciousness has returned, this objectionable feature can be avoided. While I was a member of the resident staff of Bellevue Hospital, avertin was used to produce narcosis in a large series of cases with most excellent results. The absence of nausea and vomiting, and the relaxation of the muscles that are attached to the mandible, so permitting free movement of the latter, were noted as impressive features of this anesthetic agent.

The loss of the function of mastication increases the necessity of following a carefully selected diet and of giving proper hygienic attention to the oral cavity. Liquid and semisolid foods that have a high caloric value should be chosen in order to furnish the patient with sufficient nourishment. A rubber catheter may be inserted through the nostril or mouth for

the purpose of conveying food to the stomach, but in the average case, no special means of administration are necessary. The prophylactic care of the soft tissues and fixation splints is a factor that has an important bearing on the ultimate result. A mild solution of tincture of iodine on cottor swabs should be applied to the teeth daily, and the oral cavity should be irrigated frequently in order to combat sepsis.

The usual period of immobilization in an uncomplicated fracture of the jaw is of 4 to 5 weeks' duration.

COMPLICATIONS

The late complications are cellulitis, osteomyelitis, malunion and nonunion. If swelling accompanies a fracture, it may subside after reduction, but occasionally the process goes on to suppuration, in which case incision of the external and intraoral tissues is usually necessary. If osteomyelitis occurs, ample drainage should be established and maintained, and sequestra should be removed when definitely separated from live bone. Delayed union may be from lack of early fixation, incorrect approximation of the fragments, the interposition of muscle fibers, local infection, the presence of a foreign body, and such general diseases as syphilis and tuberculosis. Fortunately nonunion is rare. If an appreciable amount of bone is lost, it is necessary to resort to bone grafting.

CONCLUSIONS

(1) Fractures of the mandible and maxilla are quite common, the former being more frequent.

(2) Proper alignment of the fragments and restoration of the normal occlusion of the teeth are the basic principles of treatment.

(3) Fractures of the condyle can be satisfactorily treated without employing an open operation.

(4) The maintenance of ample drainage in cases of a complicating osteomyelitis is essential.

(5) Careful selection of the diet and proper attention to the hygiene of the oral cavity are important factors in obtaining good results.

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NEW ENGLAND SURGICAL SOCIETY

IRREDUCIBLE DISLOCATION OF THE ANKLE*

BY ROBERT L. MAYNARD, M.D.†

ON April 28, 1936, I was called in consultation to the Barre (Vt.) City Hospital to see an anterior dislocation of the right ankle, which was of 2 weeks' standing and which was apparently irreducible. This patient was a quarry worker, 46 years old, who had been injured April 14, 1936. The history was that while he was working as a dump-man in a grout car, three or four large stones had failed to leave the car with the rest of the contents, when it was tilted sidewise. The patient stepped in front of these stones and attempted to pry them out of the car with a bar. When the largest stone started it rolled onto his right foot, hit a glancing blow on the posterior and inner surfaces, and then rolled onto the dorsum of the foot. He threw himself around and caught hold of the end of the car to avoid being carried down the dump with the stones. His foot was cut on the inside of the heel and also on the dorsum. He was taken to the hospital, and examination showed an anterior dislocation of the ankle without any evidence of fracture. A closed reduction was attempted unsuccessfully under general anesthesia. A Steinmann pin was put through the os calcis, and traction was applied for 6 days, after which another attempt at closed reduction under general anesthesia was made. This attempt was also unsuccessful.

When I saw the patient, the ankle was still in the anterior dislocated position. The patient had some motion of the ankle joint, but it was noticed that abduction was very weak, and I could not feel any contraction of the peroneus tendons when the patient attempted to move the ankle joint. I made the diagnosis of an irreducible anterior dislocation of the ankle, with pinching of the peroneus tendons between the external malleolus and the astragalus. An open reduction was done, and this diagnosis was substantiated. As soon as the peroneus tendons were lifted out of their position, the dislocation was quite readily reduced. No evidence of a fracture was found, but there was considerable thickening, and apparently an unresolved exudate around and in front of the tendo achillis. As there was a tendency for the peroneus tendons to slip, they were held back by a single strand of catgut, and the wound was closed. The foot and leg were placed in a cast for 6 weeks, after which the patient began to walk with the aid of a crutch.

*Read before the New England Surgical Society at Bridgeport, Conn., September 26, 1936.

†Maynard, Robert L.—Assistant professor of orthopedic surgery, University of Vermont College of Medicine. For record and address of author see "This Week's Issue," page 438.

There has been steady improvement, since the operation, but some swelling remains around the ankle and around the tendo achillis. The motion of the ankle is still rather limited, and

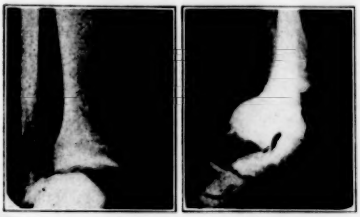

FIG. 1. Dislocation of ankle before reduction.

FIG. 2. Dislocation reduced.

the patient is not able to get his heel flat on the floor when he steps, the distance of the heel from the floor being about one third of an inch. The foot is in very slight varus. There is a depression above and in front of the outer malleolus, and the scar of operation is adherent. There is considerable atrophy, as yet, of the leg muscles. The function of the ankle joint is improving slowly, and the patient returned to work some time ago.

X-rays subsequent to reduction show bony changes taking place in front of the tendo achillis. This process does not appear to be attached to bone either above or below, and I believe it is from invasion of the exudate by bone cells in much the same way as occurs in myositis ossificans. (See fig. 4.) These bone cells were perhaps liberated at the time of the accident, when the early reductions were attempted or when the pin was passed through the os calcis. Whether this process will, in time, interfere with joint function, become absorbed, or have to be removed, remains to be seen. I do not wish to

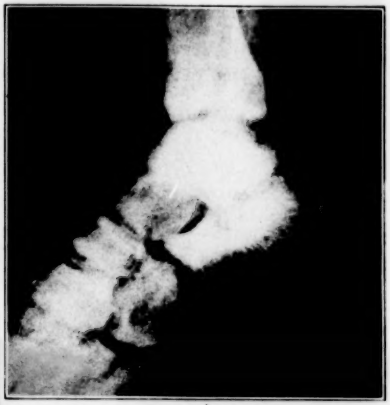

FIG 3. Follow-up x-ray, 2 months later.

interfere until it has reached its maximum, and if function continues to improve, it may not be necessary to interfere at all.

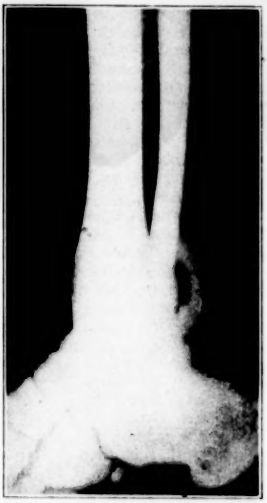

FIG. 4. Shows bony changes taking place in front of tendo achillis.

NOTE: This patient was again seen by me in February, 1937. The heel could not be brought down to the floor, and there was considerable pain in the foot. I operated and removed the bony deposit in front of the tendo achillis and lengthened the tendon. The heel could still not be brought down, so I was forced to do an astragalectomy because of the great number of adhesions between the astragalus and the tibia and fibula.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23101

PRESENTATION OF CASE

First Admission. A 40 year old Italian housewife was admitted complaining of difficulty in breathing.

Eleven years before entry the patient had an attack of pneumonia from which she recovered completely, although there continued to be slight shortness of breath. During the succeeding years she was unusually susceptible to colds, and each upper respiratory infection caused considerable increase in breathlessness. In the intervals between colds there was no cough, but there were occasional attacks of joint pain with no local redness or swelling. Four years before admission she noticed a slowly progressive weight loss associated with increasing weakness and breathlessness. Two years later, while shopping, she fainted in the street. She was carried home but was unable to lie flat in bed because of cough and a choking sensation in her chest. At that time she remained inactive in a chair for about 5 days and then was able to get about the house. Dyspnea on exertion became much worse after this attack, and she noticed evening swelling of her ankles. In addition she required two pillows in order to rest comfortably at night. The colds became more frequent, and she continued to lose strength. About 3 weeks before entry, while performing heavy housework, she had a severe spell of coughing during which she expectorated a small amount of blood. A week later she again caught cold and as usual there was marked increase in her symptoms. Orthopnea became so pronounced that she was compelled to sleep sitting in a chair. There were severe cough and dyspnea, but no further hemoptysis. Despite limitation of activities, the symptoms became progressively worse and she entered the hospital. During her illness she frequently had chilly sensations, but no well-defined rigor. From time to time there were transient pains in her fingertips, concerning which no further details were noted.

The past history is noncontributory.

Physical examination showed a well-developed and nourished woman, sitting upright in bed. There were moderate respiratory difficulty and slight cyanosis of the lips and nailbeds. The heart was markedly enlarged to the left, the maximal point of cardiac impulse being in the sixth interspace, 12.5 centimeters from the midsternal line. There was also slight enlargement to the right. The rhythm was regular. The sounds were of good intensity. There was a harsh systolic murmur at the apex, with a low-pitched, rumbling presystolic murmur in the same region. A high-pitched early diastolic murmur was heard along the left border of the sternum. Percussion of the chest showed impaired resonance at both bases posteriorly. The breath sounds were asthmatic in character, and there were numerous coarse, dry and moist rales audible generally. The spleen was palpable two fingers beneath the costal margin, but the liver was not felt.

The temperature was 101.5°F., the pulse 80. The respirations were 35.

Examination of the urine showed a specific gravity of 1.026. There was no albumin. The sediment contained occasional red and white blood cells but was otherwise negative. The blood showed a red cell count of 4,400,000, with a hemoglobin of 80 per cent. The white cell count was 13,900, 69 per cent of which were polymorphonuclears. The sputum was negative for tubercle bacilli. A stool examination, a blood Hinton test and two blood cultures were negative. The vital capacity was 1,200 cubic centimeters. The sedimentation rate was 1.98 millimeters a minute.

X-ray examination showed that the heart shadow was increased in size, the enlargement chiefly affecting the left ventricle and auricle. The aortic knob was not seen to the left of the spine. Fluoroscopy showed fairly good movement of the diaphragm on both sides, but the outlines were slightly hazy. The heart shadow was wide across the auricles. There was marked increase in size and density of the hilar shadows, especially on the right. There was general thickening of the larger lung markings, most pronounced in the right lower lung field. A film of the sinuses showed dulness obliterating the left antrum, and a similar but less marked process on the right side. There was also increased density in the region of the ethmoids and frontal sinuses.

During the succeeding 3 weeks the patient received digitalis and potassium iodide. The temperature continued to fluctuate, up to 101°F. Her cough, which continued, was productive of moderate amounts of foul sputum. At intervals she complained of pain over the left antrum, and tenderness was elicited in this region, but her condition gradually improved. A chest film was repeated on the seventeenth hospital

day. The appearance of the heart was not changed. The hilar shadows were about the same, but there was slight increase in the dulness in the right lower lung field, with some increased haziness in the right middle lung field. Subsequently the improvement continued, the cough and expectoration ceased and the temperature returned to normal. On the day of discharge another chest film showed that the heart was slightly smaller than at the previous examination, but the appearance of the lung was not changed appreciably. She was discharged improved on the thirty-fifth hospital day.

Final Admission (5 days later). Following her discharge she felt well for a day, and then began to cough and raise small amounts of whitish sputum. This condition gradually became worse and was associated with severe headache and orthopnea.

Physical examination showed an acutely ill, orthopneic patient, coughing frequently. The lips and fingers were cyanotic. The extremities were cold, but no edema was noted. The heart findings were essentially unchanged. The rhythm was regular, and the first sound at the apex was snapping. The apical presystolic murmur was accompanied by a thrill. The blood pressure was 120/80. Coarse moist rales were audible throughout both chests, and there were numerous inspiratory and expiratory squeaks. The liver edge was sharp and extended three fingers below the costal margin. The splenic edge was still palpable two fingers beneath the costal margin.

The temperature was 103°F., the pulse 120. The respirations were 35.

Examination of the urine showed a specific gravity of 1.028, with a trace of albumin. The sediment was similar to that noted on the previous entry. The blood showed a red cell count of 4,600,000, with a hemoglobin of 108 per cent. The white cell count was 25,600, 91 per cent polymorphonuclears. The sedimentation rate was 1.52 millimeters per minute.

X-ray examination of the chest showed that the consolidation in the right middle lung field had subsided slightly, but there was evidence of consolidation in the right upper lung field that had increased since the previous observation.

The patient was treated by complete bed rest, and although the lungs appeared to be clearer the other symptoms and signs remained unchanged. Her temperature fluctuated irregularly between 99° and 105°F. On the fourth hospital day, following the use of a bedpan, the patient suddenly became very restless and dyspneic. She was given an opiate which caused her to become quiet, although her pulse remained weak and the inspirations labored. Her condition became rapidly worse and she died on the fifth hospital day, 45 days after the initial entry.

Notes on the History

Dr. T. Duckett Jones: We have a history dating back 11 years with decrease of cardiac reserve preceded by acute infection with pneumonia.

In addition, there is a rather vague 11 year story of frequent colds. The increase in shortness of breath can be interpreted as a decrease in cardiac reserve, which was slowly advancing. If there had been definite episodes of illness, the history would be much more distinct as regards rheumatic fever. I should think that, if this patient had been studied or had been under medical observation during that period, evidence of active rheumatic fever would have been found, in view of the joint pain and because of the evidence of increasing heart disease. Her heart symptoms evidently had become more distinct 4 years before the fatal issue.

In regard to the episode of fainting on the street 2 years before entry, it is impossible to tell whether the onset of an arrhythmia was responsible, whether she had pulmonary edema with left ventricular failure, or whether she might possibly have had a pulmonary infaret, although the latter sounds unlikely. There was a very striking and definite increase in cardiac symptoms dating from the onset of this fainting spell.

I would assume from the story of hemoptysis 3 weeks prior to entry that the patient had mitral stenosis. Many observers of rheumatic heart disease feel that this hemoptysis is the result of the stenosis and of changes in the pulmonary circulation. In our series of rheumatic heart disease cases, we have been impressed with the fact that hemoptysis usually occurs when there is some evidence of active rheumatic fever.

Pain in the fingertips is not very common in rheumatic fever subjects. It is more apt to occur in rheumatoid arthritis, but the evidence of increasing heart disease associated with these pains and the decrease in heart reserve make one think she must at times have had active rheumatic fever.

Evidently there was no story of frank rheumatic fever at any time during the patient's past.

"There was a harsh systolic murmur at the apex with a low-pitched, rumbling presystolic murmur in the same region." This corroborates the impression that the patient had rheumatic heart disease with mitral stenosis, which had developed rather insidiously and without acute rheumatic fever.

"A high-pitched, early diastolic murmur was heard. . . ." We have, then, a patient with definite mitral disease and also some evidence of aortic disease, although there were no peripheral findings suggestive of free aortic regurgitation. The aortic disease must have been relatively mild.

It is surprising that the liver was not felt but the spleen was. The patient at that time presented a picture of failure, without the liver being down. I should think that bacterial endocarditis would have been one of the first diagnoses considered, chiefly because of her age, the findings in her heart, the fever and the enlarged spleen. She did have, however, unquestionable breaks in compensation and definite mild failure.

The urinary findings fit with either active rheumatic fever or bacterial endocarditis.

The blood findings are distinctly against bacterial endocarditis. The negative blood cultures also are against bacterial endocarditis.

I think we almost never see a more rapid sedimentation rate than this, and it is perfectly good evidence of severe infection of some type.

The lung findings are perfectly in keeping with ordinary congestion.

I do not know whether the roentgenologists would agree with me in considering all of these lung findings on the first examination as being probably due to congestion. There was a tremendously enlarged heart, not typically of a mitral shape, with prominence in the region of the left ventricle. It looks as if the aortic disease factor were more important than the physical examination led one to believe.

There is, of course, a question as to whether the pulmonary markings may not have been the result of secondary pulmonary involvement as the result of sinus infection. The ethmoid and frontal sinuses were involved. I would assume, in view of the distinct evidence of congestive heart failure, that the foul sputum was probably the result of her sinus infection, and not of lung abscess or bronchiectasis.

The second chest film shows distinct haziness, which evidently was considered to represent consolidation. There certainly is a tremendous change between the chest films taken on admission and 17 days later. This, of course, brings up the question as to whether a pulmonary infection, probably secondary to the sinus infection, has not come into the picture and is playing a pronounced part in keeping the disease syndrome going and is responsible for the symptomatology. I do not believe that from the film as presented a diagnosis of pneumonia of the right middle lobe could be made, despite the increase in markings and increased haziness throughout this area.

We have a patient whose relatively acute illness with increasing signs of left ventricular failure was present 3 weeks before admission, and she was in the hospital for 35 days, a period of 8 weeks altogether. She was evidently in fairly good condition at the time of discharge, but there is suggestive evidence that the patient was having a cyclic type of rheumatic fever, the cycles running from 4 to 6 weeks.

Upon the final admission there was an enlarged liver in addition to the palpable spleen.

X-ray plates at this time showed a good deal of increase in haziness and a good many changes in the right upper lobe.

Differential Diagnosis

In considering the differential diagnosis, I think it is perfectly evident that this patient had rheumatic heart disease for a period of 11 years and probably longer. The story of beginning dyspnea, following infection 11 years before admission, is suggestive of the silent development of mitral disease after adult life has been reached. The majority of individuals who develop silent mitral disease in early life do so at a very rapid rate and as a rule have symptoms quite early in the disease. This patient unquestionably had active low-grade rheumatic fever in the 11 years preceding admission to the hospital. For 4 years before admission she had a decided decrease in cardiac reserve and for 2 years it was so pronounced that her usual activities were considerably interfered with. For a period of 8 or 9 weeks prior to death she was much worse, had definite orthopnea, began to run a temperature and had evidence of pulmonary changes. She certainly had congestive heart failure, and I believe it likely that the patient had rheumatic fever with congestive heart failure and that the pulmonary findings must be explained on a basis that probably does not involve the sinus infection.

There are two possibilities in connection with her pulmonary changes, as far as I see them. The first is infarcts, of which this is certainly not typical. She had very little blood spitting —at least none since the beginning of the episode. She had no chest pain. The infarct or infarcts would have had to be very considerable in size, and there probably would have been some clinical episode which would have led to a diagnosis of an infarct at the time of its occurrence. The second possibility is that she might have so-called rheumatic pneumonitis. This has been described by a number of European and American observers, although there are wide discrepancies in the descriptions. She has certain features which are suggestive of rheumatic pneumonitis. One is the favorable location. Another is the clearing up of the lower part of the lung and a few days later the signs reappearing, with more definite evidence in the upper right chest. This series of events in an acutely ill person with fever makes one extremely suspicious that she might have pulmonary changes that are consistent with those seen at times in rheumatic fever. The only real feature against this is the fact that she

had very little blood spitting. Practically all of the patients I have seen with so-called rheumatic pneumonia or pneumonitis have produced a fairly large amount of blood during the acute illness. The other possibility is that this patient had a predominant pulmonary infection. Certainly there is no evidence of either lung abscess or bronchiectasis, but it is possible, I think, that she might have had a straight pneumonic process which cannot be determined from the data given us, and which I think was probably ruled out at the time when she was on the ward. It therefore seems probable to me that the pulmonary changes are related to rheumatic fever, though they do not appear to be typical.

As far as bacterial endocarditis is concerned, there are certain features distinctly against it. It is not possible to rule out acute bacterial endocarditis, but the diagnosis is unlikely. There was no bacteremia and no clubbing, which are against subacute bacterial endocarditis. The only really suggestive factors are her age, temperature and a palpable spleen. The failure to develop anemia is against it, and I think that once the rheumatic subject develops subacute bacterial endocarditis the infection generally runs a rather typical downhill course with only short remissions, and these patients are rarely discharged from a hospital in sufficiently good condition to be considered to have a favorable prognosis.

My first diagnosis, then, is rheumatic heart disease, mitral and aortic disease, congestive heart failure, and probably rheumatic pneumonitis. There was also sinus infection. The other possibilities, bacterial endocarditis and straight pulmonary infection or infarction, seem to me very much less likely. Her death was probably the result of the rheumatic fever itself.

Clinical Discussion

Dr. J. H. Means: I had this patient in my ward, and our reasoning followed much the same lines as Dr. Jones's has. We were rather intrigued with the spleen. We could not quite figure out what it signified. We thought of subacute bacterial endocarditis and thought that we had ruled it out, or at least could not rule it in. We had three negative blood cultures. There were no embolic phenomena but we were impressed with the factor of infection on her first and second entries. She had sinusitis and there was a good deal going on in her chest. There seemed to be more going on in her chest on both entries than one could quite explain on the basis of congestive failure, so that with this infectious process she puzzled us at both entries. On the first entry she made remarkable improvement. She was fretful and uncooperative on the ward and since we were assured that she would get proper care at home we let her go. There she became sick again and came back and died on the second entry. I made a note that I was much impressed with the picture of infection and thought that there was more going on than simple congestive failure. Whether it was rheumatic infection or something else, I did not know. I thought something impressive was going on in the chest, whether bronchopneumonia or multiple emboli I did not know, and I would be interested to know what the lungs showed. I did not think she had bacterial endocarditis.

Dr. Donald S. King: We saw her in the thoracic clinic before this film was taken and we voted for infarct. I do not think that we considered the rheumatic element as much as we should have.

Dr. Frederick T. Lord: With respect to infarct, it seems to me that there are objections to it in the absence of pleural pain and bloody sputum. However, it must be said that we can have large infarcts on rare instances without pleural pain.

Clinical Diagnoses

Rheumatic heart disease with mitral stenosis and regurgitation.
Cardiac decompensation.
Pulmonary infarction, multiple.
Bronchopneumonia.

Dr. T. Duckett Jones's Diagnoses

Rheumatic heart disease with mitral and aortic disease.
Cardiac decompensation.
Rheumatic fever.
Rheumatic pneumonitis?

Anatomic Diagnoses

Endocarditis, chronic rheumatic, with mitral and slight aortic stenosis.
Bronchopneumonia, atypical.
Pulmonary endarteritis.
Chronic passive congestion.
Petechial hemorrhages of the pericardium, kidneys and spleen.
Chronic fibrous pleuritis.
Arteriosclerosis, slight aortic.

Pathological Discussion

Dr. Tracy B. Mallory: Dr. Jones has put himself on fairly safe ground in raising the question of rheumatic pneumonia, because it is very questionable whether any pathologist can tell him whether he is right. This patient had, of course, an evident rheumatic heart with a fairly marked degree of mitral stenosis and quite moderate involvement of the aortic valve. There was nothing grossly or microscopically that we could feel sure was evidence of active infection. That does not rule it out, of course.

The lungs were very peculiar in gross. They were spattered with sharp, circumscribed bright red areas, which were nonrepitant, not collapsed, but not of greatly increased consistency. The gross appearance was something like infarct, something like pneumonia, and not really like either. The result was that we immediately became suspicious of the possibility of so-called rheumatic pneumonia. The only trouble is that, from the anatomic and histologic point of view, there is no good description available in the literature of what rheumatic pneumonia consists of. The clinical descriptions emphasize, as Dr. Jones has pointed out, the rapid transitory character of the lesions and the frequency of hemoptysis. I have personally seen two cases of acute rheumatism in young individuals with focal lesions throughout the lungs that suggest a partial infarction. The picture was similar to that of infarction in that there was massive hemorrhage into the alveoli, with very little lymphocytic reaction, but there was not the necrosis of the alveolar walls that one expects in a true infarct. Those cases, I think, are consistent with the typical clinical picture, but I have also seen almost every conceivable pulmonary lesion occurring in these rheumatic cases, and each lesion is almost in a group by itself. There is nothing uniform about them. This patient's lungs do not look like any I have ever seen before. There was one rather striking phenomenon which I do not think is the primary lesion, but which may have influenced the pattern of the reaction. The smaller pulmonary arteries and veins showed very extensive thickening of their walls, with narrowing of the lumina. I assume there must have been at one time a proliferative endarteritis and endophlebitis. This was so extensive and the pulmonary arteries were so markedly narrowed by it that it seems as if it must have caused severe impairment in the pulmonary circulation. Some involvement of the pulmonary arterioles is usual in long-standing mitral stenosis, but not to this degree. The next finding, which I cannot remember having seen before, was limited to the bronchial tree. Beneath the epithelium, which incidentally showed marked squamous cell metaplasia, the capillaries of the submucosa were so extraordinarily dilated that they looked almost like granulation tissue, and as one went out farther into the bronchial walls one met a great number of dilated blood vessels. These suggest rather strongly that there was a compensatory circulation arising from the bronchial arteries. It was not noted in gross that the main bronchial arteries were enlarged. We do not systematically look for them, I am afraid. I think they would have been enlarged in this case. The points of involvement in the lung parenchyma itself showed a rather disturbing picture. There was none of the acute hemorrhage into the alveoli that we would have expected in a rheumatic case, and they showed on the whole a rather ordinary looking type of pneumonia except for the fact that the alveolar walls were covered with brilliant acidophilic hyaline exudate, the type that is described in influenza, and that monocytes predominated over polymorphonuclears in the exudate. Numerous organisms compatible in appearance with pneumococci were present. Whether it has anything to do with rheumatic fever, I cannot say. What sort of picture have you, Dr. Jones, of the pathology of rheumatic pneumonia?

DR. JONES: I agree with you, that there is a good deal of confusion concerning the definite entity, not so much clinically as pathologically. From our experience and from what I have gathered from the literature, hemorrhage is probably the most distinct factor. One rarely sees the type of cell that leads one to believe it is similar to the rheumatic process seen elsewhere in the body, but it is distinctly hemorrhagic, and I have always considered this as fairly outstanding. In fact, it seems possible that the hemoptysis of mitral stenosis fits into the same category with the changes found in so-called rheumatic pneumonitis. We have general blood vessel weakness and changes, and there is a good deal of increase in pressure at various times. Why is it not possible to get hemorrhagic processes in the lung just as one observes nosebleeds and microscopic blood in the urine in rheumatic fever?

DR. MALLORY: You mean to explain the pulmonary changes as pulmonary purpura, just as you get purpura elsewhere?

DR. JONES: Perhaps it is more or less the same type of process. The fact about this case that interests me tremendously is that we have a 42 year old person dying of a disease process which we call rheumatic fever and which in young people gives a fairly distinct pathologic picture, but in these middle-aged people seems to be atypical and quite different. It is, of course, possible, as Dr. Mallory suggests, that there may have been an influenzal pneumonic process. Gross has written about the changes found in such age groups, but of course he looks for these changes extensively. I do not think we need to be worried about the fact that we do not get acute pericarditis and evidence of acute valvulitis, but we should find some changes in the heart muscle. We just discussed at the third year conference the case of a 62 year old woman dying of long drawn out infection and failing heart, who showed no acute process grossly or microscopically. If these patients have fever and clinical evidence of infection, it seems to me there should be some pathologic process to account for death, other than an old mechanical heart defect. This is

another of those cases in which it is difficult to explain the cause of death from a general routine pathological examination, omitting the possibility of influenzal pneumonia. I should like to ask Dr. Mallory if he agrees with this.

DR. MALLORY: Yes.

CASE 23102

PRESENTATION OF CASE

A 7 year old American schoolgirl was admitted complaining of abdominal pain.

The patient was well until the day before entry, when, without premonitory symptoms, within several hours she passed three watery stools stained with bright red blood. Her appetite was poor at the evening meal. Shortly afterward she developed generalized colicky abdominal pain, most severe in the region of the umbilicus, and vomited a moderate amount of clear fluid. She spent a restless night but was able to go to school on the day of entry. The abdominal pain continued, but was very much less in severity. She passed five or six loose stools, however, each of which contained blood or clots somewhat darker in color than they had been on the preceding day. Anorexia continued, but there was no further vomiting until just before coming to the hospital, when she again regurgitated clear gastric fluid. The child was put to bed and shortly afterward the parents found her lying in a pool of blood, estimated to be about a pint. This had apparently been passed by rectum.

Physical examination showed a well-developed and nourished, pale, restless, apprehensive young girl, with a sighing type of respiration. The skin was cold and moist. There was faint cyanosis of the nailbeds. The heart was normal. The blood pressure was 70/30. The lungs were clear. The abdomen was soft, and no masses were palpable. There was slight generalized tenderness, most marked in the periumbilical region. Peristaltic sounds were vigorous, but there was no evidence of fluid in the abdomen. Anoscopic examination showed a few blood clots but was otherwise negative.

The temperature was 99.6°F., the pulse 120. The respirations were 28.

Examination of the blood showed a red cell count of 3,100,000, with a hemoglobin of 50 per cent. The white cell count was 6,400, 61 per cent polymorphonuclears; 29, lymphocytes; 8, monocytes; 1, eosinophils and 1, basophils.

Shortly after entry a laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. LEO B. BURGIN: The problem in this case is essentially to explain the cause of abdominal pain and melena in a child of seven. In differential diagnosis one must consider duodenal ulcer, ulcerative colitis, acute dysentery, blood dyscrasia, volvulus, rectal polyp, intussusception and Meckel's diverticulum.

Duodenal ulcer occasionally occurs in childhood, more usually in early infancy; it may be an unexpected finding at autopsy. The history is usually of epigastric distress, possibly of some vomiting at times; but in this case such a history is absent. She had very little vomiting. She had bright red blood in her stools. Occasionally with duodenal ulcer there is gross hemorrhage, with bright red blood in the stools, but usually the stools are deep red or tarry. The pain in this case was referred to the umbilicus. With duodenal ulcer one might expect it to be in the epigastrium. In this case there was tenderness about the umbilicus, but not in the epigastrium.

Ulcerative colitis rarely occurs in childhood or with such an explosive onset. One would expect mention of pus and mucus in the stools, and I am sure that proctoscopic examination would have revealed the usual findings of ulcerative colitis.

Dysentery is usually associated with leukocytosis and fever. Here, the white cell count was normal and there was very little fever; and again no pus and no mucus in the stools.

As for blood dyscrasias, there is nothing pointing toward a leukemia or to Henoch's purpura in particular. Henoch's purpura may give rise to abdominal pain and blood in the stool in small or large quantities. It is usually associated with typical skin manifestations. Perhaps in passing we might mention the possibility of generalized telangiectases throughout the gastrointestinal tract. How that could be determined antemortem, I do not know.

When volvulus occurs, there is usually a great deal of vomiting and distention, with no blood in the stools. After 24 hours gangrene sets in, with leukocytosis and fever, which are not present in this case.

Rectal polyp would probably be found by proctoscopic examination and would probably be associated with tenesmus rather than abdominal pain.

Intussusception is a possibility. As a rule one sees it in children under two, but it may occur at any age. It occurs more frequently in males than females. The type of pain varies, but it is usually an acute colicky pain that recurs at frequent intervals. The type of bleeding is not the type described here. One usually gets blood streaks in the stool, associated with mucus, and after a short period of time there is obstipation rather than the five or six loose stools that this child had. Furthermore, with intussusception one may often feel a tumor mass

on physical examination. This was not present in this case.

Finally, we come to Meckel's diverticulum. With this condition the symptomatology varies according to whether one has obstruction, inflammation or ulceration. With obstruction we have the typical symptoms of vomiting and distention; with inflammation, or diverticulitis, one finds a history and symptomatology somewhat similar to that of acute appendicitis—pain and vomiting which tend to be quite persistent. Usually there is no blood in the stool. The third type is that which is associated with ulcer. In Meckel's diverticulum one very frequently finds gastric and pancreatic tissue. The secretion from these tissues produces ulceration, usually in the adjacent normal ileum, that goes on to bleeding such as is described in this case. There may be gross hemorrhage such as occurred before the laparotomy, or blood clots in the stool, or just blood-streaked stools as occurred at the onset of the illness. It is possible along with an ulceration of Meckel's diverticulum for one to get intussusception as well. When this is the case, the symptoms of intussusception predominate over those of the diverticulum with ulceration. My diagnosis in this case is Meckel's diverticulum with ulcer. I believe Dr. Mallory will probably describe the presence of either gastric or pancreatic tissue in the diverticulum to account for the ulceration and bleeding.

Clinical Discussion

Dr. Tracy B. Mallory: Dr. Higgins, have you anything to add?

Dr. Harold L. Higgins: I did not see this child before she was operated on. Dr. Burgin has discussed the case very well, and I quite agree with his diagnosis of a Meckel's diverticulum with ulceration and hemorrhage. I think that the absence of mucus in the stools is of diagnostic importance. Bleeding with mucus usually indicates that the lesion is in the large bowel; bleeding without mucus points toward the small bowel.

A Physician: Is it possible that some queer tumor would do this. Does the sudden onset rule it out?

Dr. Mallory: I do not believe it would rule it out, but I think on the doctrine of frequency one would be reasonably safe in discarding it.

Clinical Diagnosis

Bleeding peptic ulcer of Meckel's diverticulum.

Dr. Leo Burgin's Diagnosis

Peptic ulcer of Meckel's diverticulum.

Pathologic Diagnosis

Peptic ulcer of Meckel's diverticulum.

Pathological Discussion

Dr. Mallory: This is a syndrome that has become so obvious during the past 5 years that one wonders how it possibly could have been missed before that time. This child had, as Dr. Burgin prophesied, a Meckel's diverticulum with gastric epithelium in it, as they so often have, and an ulcer in the mucosa immediately adjacent to the gastric epithelium. The ulcers are not found actually in the islands of gastric epithelium, but in the neighboring intestinal epithelium. In this instance the ulcer was at the base of the diverticulum, where it pocketed out from the ileum, and Dr. Pratt was uncertain as to whether he would be able to get around the base of the ulcer if he tried merely to resect the diverticulum, so he thought it wise to resect a segment of ileum as well. He took about 4 centimeters of ileum along with the diverticulum. The patient made a reasonably uneventful convalescence.

A Physician: Is this severe pain characteristic?

Dr. Higgins: The other cases that I have seen have not had much pain.

Dr. Burgin: According to the literature there is very little abdominal pain, but when present it is colicky in nature and referred to the umbilicus.

Dr. Mallory: One possibility, just as in the stomach or duodenum, is perforation of the ulcer instead of hemorrhage. On clinical grounds they might have been suspicious of perforation. It had not perforated, however.

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THE FAMILY PHYSICIAN AND THE SPECIALIST

A LETTER published in this issue of the *Journal* suggests several points that seem worthy of editorial comment.

The first is the future of the general practitioner or family physician. That he will continue to exist and thrive is unquestionable. In towns and the smaller cities he still is and always will be a necessity. In the larger cities his title may be elevated to that of internist and he may decide to side-step the practice of obstetrics and minor surgery, but he will still be the family physician. In both localities he will be called in sickness, and the disposition of the case will depend upon his ability and judgment.

The second point involves the future of the specialist. He, also, is a necessity. Medical knowledge has advanced so rapidly and extensively in the past fifty years that it is beyond the mental capacity of any one human mind to learn or to retain but a fraction of the total; the same is equally true in other professions. The charges of the specialist and the expenses entailed while under his care must be higher than when the patient is treated by the family physician—adequate preliminary training in a specialty demands lengthy postgraduate instruction; adequate conditioning calls for the treatment of many charity cases on the hospital ward; and adequate care of the private patient requires hospitalization with its facilities for better nursing and for laboratory studies.

The third point concerns the relation between the family physician and the specialist. As pointed out in the letter, the family physician can satisfactorily and adequately treat over ninety per cent of the patients whom he is called in to see. It is the remainder—the less than ten per cent— that should be seen and possibly treated by the specialist; and the most important things for the good of the patient are the ability of the family physician to recognize the case that requires such care *and* his willingness to act accordingly.

The final point has to do with the ways and means of assuring the patient that the family physician is capable of carrying out his share of responsibility. This largely depends on personality and integrity; but the keystone is *professional fitness.* As previously noted in the *Journal* (**215:** 887 [Nov. 5] 1936), keeping professionally fit is entirely dependent on the attitude of the individual physician. Whether he takes advantage of his opportunities to keep abreast of the times is problematical; but, thereon, rest his true duty, his real success and the patient's welfare.

MILITARY MEDICINE

THE period from February 12 to February 22 is annually celebrated as National Defense Week. All over the country officers in the Army Reserve Corps have brought to the attention of every thoughtful citizen the fact that all the rest of the world seems to be making extraordinary efforts for secure defence. We must not forget, but look to it, that we ourselves have adequate protection. This is a duty that is common to all of us, and doctors who took part in the Great War should consider whether they do not still have a duty to perform in passing on the torch to young doctors who know nothing of war and of the medical care of men in the mass. Quite as many fighting men can be lost to service by inadequate medical care as by combat. There should be greater interest shown in the training in military matters of medical men in the medical school, in training camps, in correspondence courses, and in lectures. The education of doctors in military medicine should not be left until an emergency arises.

As an example of what is being done, there will be a demonstration of the working of a medical regiment at the South Armory on Irvington street on Tuesday evening, March 23, at eight o'clock. Doctors are urged to go and see for themselves the amount of training that is necessary to make a good medical officer out of a good doctor.

WILLIAM WITHERING AND THE FOXGLOVE

In the midst of a life devoted to the practice of medicine, with wide excursions into botany and mineralogy, William Withering found time to publish, in 1785, under the title, "An Account of the Foxglove and Some of its Medical Uses," the results of his researches on digitalis. To his contemporaries, who had long recognized the value of his botanical work, his investigation on the effect of digitalis on the human body was incidental. They had named barium carbonate, witherite; and a genus of plants, Witheringia, but none, except a few medical colleagues, urged him to set forth his views on digitalis, although it was well known in Birmingham that he had used the drug for at least ten years prior to 1785 and that its introduction into the Edinburgh Pharmacopoeia in 1783 had led to grave misuse by others of this powerful therapeutic agent. Finally, at the behest of his friends and given the leisure during convalescence from a sharp attack of pulmonary tuberculosis, Withering reported on the use of digitalis in one hundred and sixty-three cases and gave explicit directions regarding the preparation of the drug. So exact were his words that his general principles of its use are unchanged today. After describing how to make the "beautiful green powder", he writes, "I give to adults from one to three grains of this powder twice a day. In the reduced state in which physicians generally find dropsical patients, four grains a day are sufficient."

The book with its fine colored plate, now rare and costly, has, in part, been recently reprinted, with an extensive biography of the author.* This little book, written by a student of medical history, the author of a previous publication on Jenner†, brings anew to our attention the achievement of Withering.

There is an interesting parallel between Jenner's vaccination with cowpox and Withering's use of foxglove. It is reasonably clear that cowpox protection against smallpox was widely known in Gloucestershire before Jenner's "An Inquiry" of 1798. All of his colleagues *knew of the tradition,* and similar knowledge was widespread in the dairy districts in various parts of the world. Only Jenner saw its implications. Such, indeed, was the case with digitalis. Its use in dropsy was known in household medicine many years before 1785. Withering was asked his opinion about a family recipe, kept a secret by an old woman in Shropshire, as early as 1775. He noted then that the active herb, out of twenty or more in the medicine, was foxglove. It needed a man like Withering, however, to study in a scientific manner the effects of this rural remedy and to introduce it to the pharmacopoeia. Thus discoveries that revolutionize medicine are made.

*Roddis, Louis H.: William Withering: The Introduction of Digitalis into Medical Practice. New York: Paul B. Hoeber, Inc., 1936.

†Roddis, Louis H.: Edward Jenner and the Discovery of Smallpox Vaccination. Menasha: George Banta Publishing Co., 1930.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Claiborne, T. S. M.D. University of Virginia Medical School, 1930. Assistant physician, Lahey Clinic. Address: 605 Commonwealth Avenue, Boston.

Hurxthal, L. M. M.D. Harvard Medical School, 1923. Senior physician, Lahey Clinic. Address: 605 Commonwealth Avenue, Boston. Their subject is "Results of Total Thyroidectomy in Heart Disease." Page 411.

Waite, Frederick C. Ph.D. Harvard University, 1898. Formerly, assistant professor and associate professor, Western Reserve University. Now, professor of histology and embryology, Western Reserve University. Address: Western Reserve University, Cleveland, Ohio. His subject is "The Age of Harvard Medical School in Relation to That of Other Existing Medical Schools in the United States." Page 418.

Doherty, Joseph A. D.M.D., Tufts College, 1923. Visiting oral surgeon, Carney Hospital. Assistant dental surgeon, Massachusetts General Hospital. Consulting dental surgeon, Danvers State Hospital. Address: 520 Beacon Street, Boston. His subject is "Fractures of the Mandible and Maxilla." Page 425.

Maynard, Robert L. M.D. University of Vermont College of Medicine, 1911. F.A.C.S. Assistant professor of orthopedic surgery, University of Vermont College of Medicine. Attending orthopedist, Mary Fletcher Hospital. Attending surgeon, Bishop de Goesbriand Hospital. Consulting surgeon, Fanny Allen Hospital. Member, associate staff, Copley Hospital, Morrisville, Vt. Address: 289 College Street, Burlington, Vt. His subject is "Irreducible Dislocation of the Ankle." Page 428.

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OBITUARY

ROBERT BATTEY GREENOUGH
A.B., M.D.

NOVEMBER 9, 1871-FEBRUARY 16, 1937

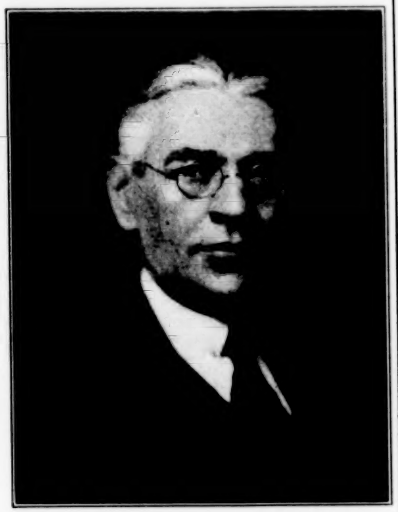

Photo by Bachrach

Robert Battey Greenough died suddenly of heart disease on February 16, 1937, in the sixty-sixth year of his life.

He was born at Cambridge, Massachusetts, November 9, 1871, the son of James Bradstreet and Mary Battey (Ketchum) Greenough.

He prepared for college at Hopkinson's School in Boston and entered Harvard receiving the degree of A.B. cum laude in 1892. He then entered the Harvard Medical School, and his work there proved a true indication of his professional career, as he graduated in 1896 with the degree of M.D. cum laude.

He was married to Amelia Mackay Goodwin at Cambridge, Massachusetts, October 16, 1900. His widow, four daughters and five grandchildren survive him.

From August 1, 1895, until December 1, 1896, he served as surgical house pupil on the East Surgical Service at the Massachusetts General Hospital and, on completing this term of service, substituted on the West Medical Service for three months.

In March, 1897, he went abroad, combining travel with study but spending most of his time in Vienna where he devoted a great part of his time to pathology. This period of study had a profound influence on his later professional career. He returned to this country and opened an office for the practice of medicine in November, 1897, becoming the private assistant of Dr. John Collins Warren.

He was appointed surgeon to outpatients at the Massachusetts General Hospital in June, 1900, and from that time he served the Hospital continuously in some capacity up until the time of his death. He became assistant visiting surgeon in 1911 and visiting surgeon in 1916. In 1932 his active service to the Hospital was terminated by the age limit and he became a member of the Board of Consultation. He was one of the prime movers in the establishment of the Tumor Clinic at the Hospital which has proved of great value both to the patients and to the staff. His attitude toward the Hospital can best be shown by the question that he was in the habit of asking his colleagues and assistants when a serious change in policy was under consideration, "What effect will this have on the Hospital?" This attitude of mind on his part had a profound influence in molding the characters of the many house officers who served under him, since loyalty to a hospital is unfortunately not always the prime factor which determines the acts of the staff.

He was appointed assistant in surgery at the Harvard Medical School in 1901, instructor in surgery in 1905 and assistant professor of surgery in 1910, holding the last position until 1932, when his retirement from active service at the Massachusetts General Hospital necessitated his retirement from the Medical School.

He was an enthusiastic teacher and won the respect and affection of many generations of medical students as a clear and inspiring lecturer, the honesty of whose teaching was apparent to all who studied under him.

For many years he served as a member of the Cancer Commission of Harvard University and for some years as its chairman. He had a major share in the organization of the Collis P. Huntington Memorial Hospital, of which he was appointed surgeon-in-chief in 1915, a position that he held for many years, becoming a member of the consulting staff on his retirement. He remained a member of the Cancer Commission up until the time of his death. In 1927 with the late Dr. George Bigelow, then health commissioner of Massachusetts, he took a leading part in the organization of the Pondville Hospital, and was largely responsible for the selection of the clinical staff. In this hospital he held the position of chief surgical consultant up to the time of his death.

In March, 1915, he went abroad with the first Harvard Unit as surgeon and executive officer, serving three months at the American Ambulance at Neuilly-sur-Seine, France. In 1916 he

joined the medical corps of the Naval Reserve, being commissioned as lieutenant, junior grade, and when this country entered the World War, he was ordered to active duty at the United States Naval Hospital, Chelsea, Massachusetts, as chief of the Surgical Service. July 28, 1917, he was enrolled as surgeon in the United States Naval Reserve Force, Class 4, with the rank of lieutenant commander. He was relieved of active duty, February 3, 1919, but was later recalled for active service for one day and was appointed and enrolled as medical inspector with the rank of commander, U. S. N. R. F., Class 4.

He then returned to private practice, devoting himself almost entirely to the surgery of cancer and allied conditions. He wrote many articles and addressed many meetings on the surgery and prevention of cancer, becoming nationally recognized as an authority. Although he made many contributions to the increase of our knowledge about cancer, perhaps his most important work was in evaluating the overenthusiastic claims of others in regard to the discoveries of the etiology and treatment of this condition. I well remember a remark he made to me after a long investigation of the claims of various writers, both as to causation and cure of cancer, which had led to purely negative results. He said after many weeks of hard work, "Well, if I have not learned anything about the etiology of cancer by this work, I have at least proved what cancer is not, and no one else will have to waste his time doing this over again."

He was extremely active in medical societies and was honored by being elected to high office in many of them. A list of the more important follows, although undoubtedly it is incomplete.

Massachusetts Medical Society.
Member of the Council for several years.
President 1929-1931.

American Surgical Society.

American College of Surgeons.
Member of the Board of Regents.
President 1934-1935.

Society of Clinical Surgeons.

New England Surgical Society.

Boston Surgical Society.
President 1928-1930.

American Association for Cancer Research.

American Society for the Control of Cancer.
Director for many years.
President 1936-1937.

American Academy of Arts and Sciences.

In the autumn of 1936 he was appointed a member of the Board of Managers of the Memorial Hospital for the Treatment of Cancer and Allied Diseases, New York City.

Although much of this work can well be classed as public service, rather than medical work, the only definite public service appointment that he received was as a member of the Medical Advisory Committee on Social Security in Washington in 1934-1935.

As a general surgeon he won comparatively little recognition, but he was recognized as one of the real authorities on cancer and its treatment, both operative and palliative.

He was perhaps the best organizer and executive the writer has ever known, and his work as secretary of the General Committee of Organization for the meeting of the American Medical Association in Boston, which came after a lapse of many years, set a standard which has never been surpassed. The recognition gained by this work led to his appointment, usually as chairman or secretary, on many important committees and much of his life was devoted to unpaid service of this nature, often with serious detriment to his private practice. How much time he spent in this work can best be appreciated in the light of a remark he once made to the writer, that he was usually so tired at night that he was unable to give adequate attention to the various surgical articles and reports which he had in process of preparation at all times. To compensate for this he began his work at five o'clock in the morning, when he could count on being clearheaded and on having two or three hours when all was quiet; he could concentrate on the problems in hand, and most of his literary work was done in these hours.

During the last few years of his life his health was far from good; he often suffered considerable pain, but he never allowed his personal disabilities to interfere with what he considered his duty. His outstanding characteristics were loyalty, courage and sacrifice of self to his ideals.

He was an excellent teacher, clear in his exposition and absolutely honest in his teachings, never using catch phrases to cloud the issue at hand and always being ready to say he did not know the answer to any question which might be under discussion. He was an inspiration to the younger men who came in contact with him and he trained a most promising group of assistants who can be depended upon to carry on his work. He was uncompromising in the stand which he took in regard to what he considered necessary reforms in medical practice, but was so fair-minded and so tactful in listening to the arguments on the other side that he usually retained as personal friends the very men whose theories he fought most bitterly.

There is no question but that the thousands of miles he traveled in his work as president of the American College of Surgeons and of the Massachusetts Medical Society, at a time when his health was far from good, seriously sapped

his vitality, especially when combined with the strain of addressing medical and public gatherings night after night. He undoubtedly would have lived years longer if he had been willing to spare himself, but he regarded every call made on him as a public duty and never learned to say, "No," or to think of himself. In his death the medical profession has lost one of its real leaders.

He had an extremely attractive personality and an unusual faculty for making and keeping friends even though his manifold activities made it impossible for him to meet them often. He had drunk deeply of the fountain of eternal youth, retaining his enthusiasms to a marked degree.

Outside of his medical and family relations he had one great hobby—fishing. As a result of many fishing trips with him, in the days before his health interfered, the writer knows that he was an extremely keen fisherman and woodsman and far more skillful than the average.

F. S. N.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

R. S. TITUS, M.D., *Chairman*
472 Commonwealth Ave., Boston, Mass.

R. J. HEFFERNAN, M.D., *Secretary*
1101 Beacon St., Brookline, Mass.

CASE HISTORIES

No. 10. HYDATIDIFORM MOLE

Mrs. M. T., a white woman thirty-two years old, was seen in the office on February 18, 1935. She had been referred by her family physician for advice as to treatment. Her last period began May 18, 1934 and was normal in character and duration. During June and July the patient complained of engorgement and tenderness of the breasts, an indifferent appetite, flatulence and an "upset feeling" of her stomach. Her basal metabolism rate on July 19 was plus 9 per cent. Urinalysis and blood examination were reported to have been normal. She gained ten pounds in weight during the summer months but observed neither enlargement of the abdomen nor fetal motion. Following a vaginal examination early in November, her physician expressed the opinion that the patient was not pregnant and prescribed a tablet containing 5 grains of ovarian extract and ¼ grain of thyroid gland to be taken three times a day. Her basal metabolism rate at the beginning of this therapy was minus 4 per cent.

On January 4, 1935 there was slight spotting of bright, normal appearing blood which lasted only a few minutes. After this flowing occurred, her physician gave her five intramuscular injections of an estrogenic hormone. On January 28, a few days after the last injection, she began to have a bright red flow which was followed by a dark-brown discharge containing small stringy blood clots. During the afternoon of February 1 she began to have crampy pain in the lower abdomen accompanied by a profuse discharge of bright red blood from the vagina. During the evening she passed several large clots and an ovoid, discrete mass which the patient described as "twice the size of a hen's egg". The following day the specimen was sent to a pathological laboratory for examination and was reported to have been a "hydatidiform mole of a nonmalignant type". Flowing continued for about a week and was followed by a slight, dirty brown vaginal discharge which was still present on February 18.

There was no family history of the major systemic diseases. Except for the less serious diseases of childhood the patient's health had always been good until 1928 when she suffered what she described as a "nervous breakdown". Her only previous pregnancy was normal throughout and was terminated in 1930 by forceps delivery of a baby weighing eight and three-quarters pounds. For several months following delivery she had profuse leukorrhea, severe sacral backache and a "dragging sensation" in the pelvis. In 1932 a dilatation and curettage, an amputation of the cervix, an appendectomy, a left oöphorectomy and an Olshausen suspension of the uterus were performed by a competent surgeon. The left ovary was found to be large and showed extensive cystic degeneration. Both tubes and the right ovary were normal. The indications for operation were: extensive laceration of the cervix with erosion, retroversion of the uterus, chronic appendicitis and inflammation of the left ovary. The pathological diagnosis was: follicular cysts of the ovary and chronic appendicitis. The patient made a good postoperative recovery and had been very well until the onset of her present illness.

On physical examination the patient was well developed and fairly well nourished. Her weight was 119 pounds. Her color was good and her skin clear. There were no palpable enlarged glands. The heart was normal in area; the sounds were normal and the rhythm regular. The blood pressure was 114 systolic and 68 diastolic. Her temperature was 98 degrees, her pulse 82 and her respirations 18. The breasts were small, flat and flabby and lacking

*A series of selected case histories by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

in mammary tissue. The nipples were normal. The abdominal walls were smooth, firm and unstriated. There was a median laparotomy scar 19 cm. long in the lower half. There were no masses on palpation, and no tenderness was noted. On vaginal examination the patient presented a small, parous orifice and an excellent perineum. The vaginal mucosa was normal in appearance. There was neither cystocele nor rectocele. The cervix was short and slightly cystic and when viewed through a bivalve speculum was found to be normal in appearance except for the scar of a low amputation. A small amount of rust-colored discharge oozed from the external os. The uterus was in anterior position, of normal size and apparently well suspended but slightly softer than normal in consistency. The adnexa were normal.

On account of the history of the recent expulsion of a hydatidiform mole, exploration of the uterine cavity for possible retained fragments was recommended. This advice was not followed until February 27 when the patient entered the hospital. Under gas-oxygen anesthesia the cervix was dilated with a Goodell dilator and the uterine cavity lightly curetted with a blunt curet. A small amount of what appeared grossly to be hyperplastic endometrium was obtained. The pathological report was as follows:

Grossly the specimen consists of uterine curettings, the entire mass measuring 18 by 12 by 3 mm. No cysts were found.

Microscopically the sections show tortuous endometrial glands lying in a stroma containing numerous inflammatory cells consisting mainly of polymorphonuclear leukocytes and round cells. The glandular epithelium shows definite proliferation with enfolding of the walls and piling up of the epithelial cells. There are numerous glands in which the epithelium appears degenerated. There are in addition areas of recent hemorrhage into the stroma. An organizing blood clot is also included in the section.

Diagnosis. Adenomatous hyperplasia of the endometrium (slight).

Following the curettage a Friedman modification of the Aschheim-Zondek test was done on March 1 and was later reported to be negative. The patient was discharged well on March 3.

Pelvic examinations and Friedman tests performed six months and one year later were both negative.

The patient reports her menstrual periods to be normal in character and rhythm.

Comment: There are several interesting features to be noted in this case. First, the size ascribed to the mole seems inconsistent with the period of amenorrhea which began on May 18, 1934 and ended with expulsion of the mole on February 2, 1935. This suggests the possibility that the absence of menstruation in the beginning may have been from a cause other than pregnancy. Secondly, the scanty flow in which no characteristic cysts were observed prior to the ultimate delivery of the mole is unusual and, no doubt, tended to obscure the diagnosis. Thirdly, the fact that this patient had cystic degeneration of the left ovary is in keeping with reported observations on association of mole with this type of ovarian disease. Fourthly, the expulsion of the mole a few days after the intramuscular injection of an estrogenic hormone, while probably merely incidental, warrants the suggestion that in cases of amenorrhea of doubtful origin all reasonable measures to exclude pregnancy as the cause should be invoked.

Although the use of a curet in a uterus which harbors or has recently harbored a mole is fraught with risk of perforation, it seemed advisable to explore the uterine cavity in view of the confusion of observation and diagnosis in this case.

It is recommended by eminent authorities that cases of hydatidiform mole continue under observation for at least 2 years after the expulsion of the mole and that Aschheim-Zondek or similar tests be performed at least once a month for 1 year, to rule out the possible development of chorio-epithelioma.

OMISSION FROM DIRECTORY

Through an error the name of Robert Earle Glendy, 219 Commonwealth Avenue, Newton (office 375 Commonwealth Avenue, Boston), was omitted from the Annual Directory.

MISCELLANY

ADDRESS BY DR. MORRIS FISHBEIN

Dr. Morris Fishbein, Editor of the *Journal of the American Medical Association*, spoke on "The Frontiers of Medicine" at the old South Forum recently. Among the topics he discussed were the problems facing modern medicine in the line of social legislation and preventive medicine, and the problem of medical quackery.

THE ST. LOUIS MEDICAL SOCIETY

The St. Louis Medical Society has recently issued invitations to the commemoratory observance of its one-hundredth anniversary, which will be held from April 5 to April 7.

THE PHARMACOPOEIA AND THE PHYSICIAN

The series of twenty-four articles now appearing in the *Journal of the A. M. A.*, dealing with the use of

official medicines in medical practice, has created a very wide interest in both pharmaceutical and medical circles. These articles, written by eminent clinicians, and developed through the cooperation of a U.S.P. committee of physicians and pharmacists, working with the officials of the *Journal of the A. M. A.*, have suggested the proper and most effective therapeutic agents indicated for a wide variety of treatments.

This series, covering as it does, suggestions for prescribing in a broad field of therapeutics, will prove a valuable addition to the library of the physician and the pharmacist.

CORRESPONDENCE

THE FAMILY PHYSICIAN AND THE SPECIALIST

January 28, 1937.

Editor, *New England Journal of Medicine.*

In these days of great anxiety and uncertainty regarding the future of medicine, the way lies, not in a *turn* toward or away from state medicine but in a *return* to the general practitioner.

The romantic halo that unwise publicity has placed around disease and specialists is fast pushing the general public into a state of voodooism. The fetish is the specialist and his doings are magic.

The economic result of this is apparent. I had a case about a month ago, which illustrates the point. A two and one-half year old female child contracted bilateral bronchopneumonia. The parents forced me to call in a pediatrician. The child was hospitalized, had day and night nurses, was x-rayed, was placed in an oxygen tent, and so forth. In 5 days her pneumonia was better. Two days later she developed a left otitis media, and the pediatrician called in a brother specialist, the ear man, who did a paracentesis to the tune of fifteen dollars. The child remained in the hospital for 2 whole weeks and is now convalescing under the care of specialists and nurses.

Bronchopneumonia in a robust child of that age, when complete resolution is attained in 5 days, can be treated satisfactorily by any good general man without the help of nurses and what not. The paracentesis would have been done as a mere routine procedure during a house call. This sickness will cost the parents a good two hundred dollars. It would have cost them about forty dollars under the general practitioner's care.

It has been said before that 90 per cent of sickness can be handled properly by the competent general man. Most general practitioners are competent and striving hard to better themselves. They would be greatly helped along this line if the hospitals cooperated a little more.

Modern medicine is based on the autopsy and the study of both gross and microscopic pathology. Modern surgery, which, in the living frequently furnishes facts that are as valuable as those at the autopsy, has also helped a lot in sharpening our diagnostic acumen. Since the findings of both are so valuable and since early diagnosis of cases, by far the hardest to make, is and will ever be the responsibility of the general practitioner, why not give him the valuable help afforded by those few methods?

It would be a small matter to typewrite routinely the operative note of the surgeon and the autopsy protocol, if any, in duplicate and to forward a copy to the general man, who referred the case into the hospital. In that way he would be able to check his diagnosis and to mend his methods accordingly. The exact state of affairs, as found at operation, would also help in the prognosis and after-care of the patient.

I believe that, if you initiated something toward having this come about, most general practitioners would be greatly benefited and grateful.

ROMEO J. COURNOYER, M.D.
40 Cherry Street,
Spencer, Mass.

EDITOR'S NOTE

The correspondent's point in regard to copies of operative notes and autopsy protocols is well taken, but is already provided for by many hospitals. The majority of the larger hospitals in Boston forward some data on each case to the referring physician, and more detailed information is available for the asking. The completeness of the data received by the referring physician seems to depend, to a large extent, upon his initiative.

STATE MEDICINE

February 22, 1937.

Editor, *New England Journal of Medicine,*

I hope that every doctor in the State has read the article "Insurance of Health Urged" by Robert L. Norton, in *The Boston Sunday Post*, February 14, 1937. This article is certainly a piece of paid propaganda by a paid commentator who apparently does not know what he is talking about. Some of the conclusions he draws are correct, but ninety per cent of them are wrong. This is the same writer who, at election time, came out with a big story about the Federal Reserve System and its wonderful attributes, but he clouded the story so much that the ordinary person believed that he was right, when he failed to give the correct story on the once defunct Federal Reserve Banks.

He wants to take the practice of medicine out of the doctor's hands, because there are, according to him, eighty million people in this country who either do not receive the care they need or are heavily burdened by its costs. The doctors know this is not the truth.

We will pass over the fee-splitting part, with the remarks that this is an insignificant consideration. His description of a successful doctor is ludicrous, but it makes good copy in the news or movies. The people know that their doctor is a hard-working, conscientious man, who does not comply with Mr. Norton's description.

To continue, he quotes from a recent survey of

the Julius Rosenwald Foundation, a report that the medical profession knows does not give the views of the doctors of the country, but the views of biased investigators. Here are some of the cases:

1. Patient, aged 57, meat dresser. Fractured skull, question permanent handicap. No savings. Urged to go into the wards of the Massachusetts General Hospital. Children working and wished him to go to the Baker, and so forth. Bill of $971 paid before discharge by married children.

What is wrong with that picture? Was he hit by an automobile, or was this a compensation case? If so, how much did the children collect? He was urged to go into the wards of the Massachusetts General Hospital, but the children wanted him to go to the Baker. If they wanted private treatment they could afford it, just the same as if they want a box seat at the theater they pay for it.

2. About the same. Unemployed, but sister wanted him to keep out of the wards at the Massachusetts General Hospital, so the bill was $619.

If the sister wanted him to go to a private hospital she surely could afford such treatment.

3. A college professor making $4000 a year could not afford to pay $150 for a tonsil operation, so he paid $25 and got a $25 job.

A man making $4000 per year can certainly pay $150 for an operation. I will wager that he has a medium-priced automobile and that he has a substantial home; and if such a case is typical of this Julius Rosenwald Foundation, one can surmise what is in back of such a foundation.

I have read over that most enlightening pamphlet, "On the Witness Stand", and would suggest that one be sent to Mr. Norton for his perusal, before he comes out with any more of his authoritative articles about medicine and medical practice. I am very pleased to learn from this pamphlet that the solution is "an annual living wage" for the working man so that he can take care of himself, and so that all these so-called social reformers or securists can go back to their jobs of minding their own business. There is only one man who has advocated this annual living wage, year in and year out, and he also states that the country can run along if such a condition is brought about. He is back again on the radio every Sunday from three to four o'clock.

How long is such a powerful society of men and women going to remain dormant and let such paid reporters influence the thought of the people? The people are anxious and waiting for some word from the medical societies about these conditions, but nothing is coming forthwith. The Massachusetts Medical Society should not allow such propaganda to be circulated among the people. With modern leadership this Society should be all-powerful in this state, and that with a minimum cooperation by all the doctors in the state or nation. This question of state medicine will be decided by the politicians and not by the doctors, and I cannot think of a more powerful group, if they want to take it upon themselves to become politicians, than the doctors of this state. Awaken to this fact before it is too late!

JOHN V. McMACKIN, M.D.

27 Bay State Road, Boston.

THE EPIDEMIC OF SMALLPOX ON THE ISLAND OF ST. KILDA IN 1724

February 24, 1937.

Editor, *New England Journal of Medicine*,

The following account is taken from "My Joyful Life" by C. J. Cutcliffe-Hyne, Hutchinson & Company, Ltd., publishers, London:

The author tells us that Stack Borrerah is the largest of the St. Kilda group, off the coast of Scotland, and that the island was used for grazing for "a good head of sheep". The shore was very steep and rocky, and there was no suitable place for pulling up a boat, the account states, so it was necessary to ferry the sheep shearers over. They were marooned on the island until the shearing was completed. The shearers "live on mutton and puffin and fulmar" (the fulmar petrel), "living in a cave". When their task is over they light a signal fire and are called for and taken back to "the embraces of their own domestic fleas".

We are told that in the year 1724, the shearers finished their work as usual, and lighted their signal fire. No attention was paid to their signal from St. Kilda, not even to repeated signal fires. The poor shearers were obliged to stay on the island until spring, when the factor's smack came over from the mainland with supplies for the Kildians. She was sighted by the marooned shearers, and after frantic signaling, at last headed for their island, lowered a boat and took them off, then headed in for St. Kilda. It was noted that there was no smoke from the houses, and we are told no answer was made to the signal gun. The captain went ashore alone, being suspicious of some great tragedy. There was not a living soul on the island. Smallpox had carried off every inhabitant, and the savage dogs, mad with hunger, had actually left not a sign of a human being. It was found later on investigation that a small vessel, a "pinkie", which the shearers had sighted while working on Borrerah, had dropped into St. Kilda's harbor and brought the smallpox to this virgin soil, where it attacked the whole population.

The author concludes: "So the smack sailed back to Skye . . . and left St. Kilda to stew as it chose for the next fifty years. Then the Macleod of the day decided that the Western Ocean gales had scoured the island free of the old pestiferous atmosphere and recolonized it. He had a crop of criminals on his hands and nowhere else handy where they might be deported."

WILLIAM PEARCE COUES, M.D.

Brookline, Mass.

BETTER CARE NEEDED FOR THE MENTALLY ILL

Editor, *New England Journal of Medicine*,

An editorial entitled "Our Insane Population," appearing recently in *The Boston Sunday Herald* together with a letter from Henry W. Jarvis suggesting an excess of unnecessary commitments to our State institutions, opens up a vital subject which has long been in need of an airing.

Undoubtedly the Massachusetts law needs overhauling; but, as concluded in this editorial, we must avoid snap judgments if any real progress is to be made. In the meantime, while we study the laws of other states whose per capita insanity census figures are significantly lower, with a view toward amending our own statutes, it would seem logical to work now toward a more open-minded application of the laws we already have. In an age when liberal interpretation of outmoded legislation has been generally applauded, our Department of Mental Diseases clings doggedly to its letter-of-the-law interpretations with only secondary thought for the welfare of the individual patient.

The formation of sane laws to deal with insanity is tremendously difficult, since the entire problem is so largely one of degree. With the thousands who find themselves temporarily out of adjustment mentally with society it is very seldom a simple question of "yes" or "no" regarding insanity. Few legislators, and still fewer psychiatrists, would attempt to put their finger on the point where sanity leaves off and insanity begins. In most cases, therefore, no actual definition of insanity is called into play. It is up to the two examining physicians to decide merely whether the patient's degree of maladjustment is sufficient to make commitment appear advisable.

Admittedly then, since — as leading psychiatrists will readily admit—what looks like a fair judgment of one's behavior pattern today may be proved wrong tomorrow, there is bound to be a wide range of error, and perhaps a certain amount of abuse. On the other hand it is equally true that, owing to the frequency of real emergency conditions, it is wise to continue within the law some process which makes it possible to "put away" quickly an individual whose sudden maladjustment is expressed in actions dangerous both to himself and to those around him.

One suggestion which, it seems to me, might go a long way toward eliminating the abuses suggested in the Jarvis letter would be to place all commitment procedure on the "30-day paper" basis, with certain additional safeguards for the patient's protection. In other words, make it obligatory that any individual whom two or more physicians agree is "eligible" for commitment be granted a generous period of observation in some State or private institution *before* final judgment as to insanity is passed. Furthermore—and this is equally important if we are to defeat the railroading tactics of scheming relatives—why not have it written into the law that a final judgment on extended commitment can be taken only after three specialists in the State's employ have reviewed and approved all "evidence" compiled by local staffs, and have interviewed the patient, regardless of whether said patient is "on trial" in a State or a private institution?

While a procedure of this kind would entail the expense of several roving commissions, it seems likely that the expense might ultimately become a profitable investment, both in human values and in dollars. While it would tend to prevent unnecessarily extended commitments through collusion, it might also reduce the number of mild "borderline" cases in overcrowded State institutions by fostering an active system of "parole" to approved private establishments.

Regardless of whether the above suggestions are worthy of consideration, two points remain undeniably true: (1) To handle adequately the problem of insanity we must have *some* kind of law; and (2) whether we continue with the present statute, or seek amendment of it, a *liberal interpretation* of said law should be demanded at all times. The law we must have as a guidepost from which to chart the courses of those suffering from mental illness. But, to cling stubbornly to the precise lettering on this guidepost, regardless of its effect upon the welfare of the individual patient, often works more harm than good and makes for anything but progress. I am not alone in my belief that our Department officials have too often fallen into the rut of interpreting the law for its own sake rather than for the sake of those it was intended to serve and protect. It is so much simpler that way! Yet the Department officials have always known—as have all psychiatrists, psychologists, general medical practitioners and thousands of laymen—that the traffic of mental disease is strictly a traffic of individuals with individual problems demanding individual solutions. It never can be handled successfully with a red and green light system such as takes care of automobile traffic!

Many nervous patients of the "borderline" type, for example, while "technically committable" in the eyes of the Department and therefore not legally to be cared for in unlicensed institutions, actually could—and often do—receive a far higher grade of individual care at small private establishments of the more informal type. Sanitaria of this kind can offer the patient a normal, homelike existence, usually under medical supervision fostering helpful occupational outlets, and quite free from depressing association with the obviously insane or feeble-minded. Good food and nursing care are assured by the simple fact that the operators of such organizations are bound to do a good job or lose their source of livelihood, for the patient is not required to stay. It's the age-old story of the satisfied customer passing around the good word.

Obviously in such natural, unregimented surroundings, the individual's chance of recovery is, in the majority of cases, greatly enhanced. And, as indi-

cated earlier, a more extensive use of such facilities —which are plentiful in Massachusetts—would tend to relieve the pressure of committed cases on our State institutions. Surely it is not unreasonable to presume that many "doubtful" patients, granted an extended time (by a liberalized law) in which to attempt a personal readjustment under the more normal social conditions just mentioned, would ultimately "beat the rap" . . . at a considerable saving to the State.

While there is no doubt considerable scheming among "cruel and selfish relatives" eager to transfer their burdens to the State via the commitment method, my experience has convinced me that much of what is hastily assumed to be dark plotting is merely the result of fear, ignorance and the uncompromising application of the laws relating to insanity.

Let us consider, by way of example, a typical predicament faced by a family of very moderate means. Grandmother, who has lived with them for a number of years, gradually drifts into a state of senility. Her memory of current happenings and associations has become vague, almost useless. There is the constant danger that she may become seriously injured in a fall, or wander from the home and not find her way back, thus making it obligatory that some member of the family be with her nearly every moment, day and night. Add to this the fear that, at any moment grandmother may become utterly unmanageable and it is easy to understand the feeling of relief experienced by her relatives when the physician advises them that grandmother is a "fit subject" for commitment.

It turns out, however, that this is only the beginning of the family's dilemma. They discover that if they go through with a commitment they are faced with only two alternatives, one of which is impossible and the other distasteful. On the one hand they find the licensed private institutions offering individual care for committed, or committable, mental patients, with staff and equipment costs accounting for a weekly guest rate far in excess of their means. On the other hand they find the large state institutions, only nominal in expense, but so overcrowded that it would be impossible for grandmother to enjoy any semblance of the home life to which she had been accustomed. Between the two extremes they may discover many nursing homes and small unlicensed sanitaria, obviously well equipped to care for grandmother, individually, at moderate rates . . . if she is not committed. When they are advised, however, that the mere fact that grandmother is legally committable (even though in no way dangerous) denies to her the privilege of such care, they are about ready to give up in despair. After struggling along for a few weeks more at home, they finally take the inevitable course and turn grandmother over to the State.

After all is said and done, isn't it high time that our law was humanized sufficiently to allow our small private institutions to function to their fullest capacity in service to the State as a whole, not alone as havens for the confused aged but also as "preventoriums" against the onset of serious mental illness among younger members of society? Let them be assigned specially restricted licenses, let them be inspected by State authorities at regular intervals. . . . But, whatever the system ultimately devised, let us delay no longer in putting to better use these advantages we already possess!

F. WALLACE PATCH.

Framingham.

RECENT DEATHS

LAWRENCE—JAMES WILMOT LAWRENCE, M.D., of 25 Washington Street, Malden, died suddenly March 5, aboard the steamer, Lady Rodney, while returning from a trip to Jamaica. He was in his seventy-fifth year.

A native of New Brunswick, Dr. Lawrence attended McGill University, and was graduated from the McGill University Faculty of Medicine in 1893. Before coming to Malden, he served as superintendent of Winnipeg Hospital, Winnipeg, Ontario. He had practiced medicine in Malden for more than forty-three years and was a member of the staff of the Malden Hospital.

He was a Fellow of the Massachusetts Medical Society and the American Medical Association.

PRIOR—EARLE FRANCIS PRIOR, M.D., of 52 Main Street, Cochituate, died on March 4, 1937. He was in his fortieth year.

A graduate of Tufts College Medical School, Dr. Prior was at one time city physician in Malden and did considerable work for the White Fund Clinics in Boston.

He was a member of the Massachusetts Medical Society and the American Medical Association, and of the staff of the Malden Hospital.

OBITUARY

DR. SYLVESTER F. McKEEN

By the death of Dr. Sylvester Forshay McKeen on November 29, 1936, the medical profession lost an outstanding general practitioner.

Dr. McKeen was born in Bridgewater, N. S., November 3, 1873. After spending one year at the University of New Brunswick, he transferred to Harvard College. Because of ill health he had to give up his work there, and went to Colorado for a year. He did not finish his college course, but entered Harvard Medical School the next year, graduating therefrom in 1896. Following a year's internship at the Boston City Hospital he went to Vienna for study. On return from abroad he settled in Allston for the general practice of medicine. After twenty-five years he moved to Brookline.

He was a valued member of the Massachusetts Medical Society and of his district society. He was a censor of the Norfolk District Medical Society from 1926 to 1931, vice-president of the Norfolk District in 1928 and its president in 1929, and then a member

of its nominating committee. In 1923 he was appointed a member of the Committee on Ethics and Discipline of the Massachusetts Medical Society, and continued as a member of that committee until he was relieved from service in June, 1936, because of his serious illness. He served as a councilor of the Massachusetts Medical Society from 1926 to 1935.

Uncomplaining and cheerful even after he well knew that he had but a short time to live, he kept a keen interest in the activities of the Massachusetts Medical Society and was most interested in the welfare of his patients. He was quiet, dignified, devoted to his profession and to his patients, a man of few words and of great gentleness. His voice was always on the side of kindliness and justice. His death is a great loss to those of us who have served on committees with him and have been associated with him in practice.

In 1900 he married Teresa Foster. She and their three children survive him.

NOTICES

EXAMINATION FOR MEDICAL ADVISER TO INDUSTRIAL ACCIDENT BOARD

Thomas H. Green, state commissioner for civil service, announced recently that the position of medical adviser to the state industrial accident board will be offered to Massachusetts physicians through a competitive civil service examination. Commissioner Green is ordering the examination in line with Governor Hurley's drive to fill positions in the public service through competitive examinations.

The position has been held since January 24, 1937, by Dr. Matthew V. Norton of Jamaica Plain, who was appointed by the industrial accident board to fill the vacancy caused by the death of Dr. Francis D. Donoghue. Dr. Norton will be among the many physicians who are expected to compete for the appointment.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, March 18, in the Amphitheater of the Peter Bent Brigham Hospital, Dr. Reginald Fitz, Wade Professor of Medicine, Boston University School of Medicine and Director, Evans Memorial, will give a medical clinic. Practitioners and medical students are cordially invited to attend.

BOSTON PATHOLOGICAL SOCIETY

The next meeting of the Boston Pathological Society will be held Friday, March 19, at 8 p. m. in the Amphitheater (Vila Street entrance) of the Children's Hospital.

PROGRAM

Processes Antecedent to Tumor Formation Induced by Certain Carcinogenic Chemicals. Dr. S. B. Wolbach. Physicians and medical students are cordially invited.

S. C. DALRYMPLE, M.D., *Secretary.*

SUFFOLK DISTRICT MEDICAL SOCIETY

8 Fenway
Wednesday, March 31, 1937, at 8:15 p. m.
At the Boston Medical Library

Social Insurance—It Affects the Medical Profession. Charles E. Mongan, M.D.
Discussion: Channing Frothingham, M.D.
Refreshments!

CHARLES C. LUND, M.D., *Secretary.*

BOSTON MEDICAL HISTORY CLUB

8 Fenway
Monday, March 15, 1937, at 8:15 p. m.
At the Boston Medical Library

Medical Service During the Siege of Boston. A. Warren Stearns, M.D.
Illustrated by lantern slides.

BENJAMIN SPECTOR, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheater (Shattuck Street entrance), Tuesday evening, March 23, at 8:15 p. m.

PROGRAM

Presentation of Cases.

Hyperinsulinism. Allen O. Whipple, M.D., Valentine Mott Professor of Surgery, Columbia University College of Physicians and Surgeons, New York City.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MARCH 15

Monday, March 15—

8:15 p. m. Boston Medical History Club. Boston Medical Library, 8 Fenway.

Tuesday, March 16—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Some Aspects of the Treatment of Lobar Pneumonia. Dr. Maxwell Finland.

*12 m. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

Wednesday, March 17—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

†12 m. Clinicopathological Conference. Children's Hospital Amphitheater.

4 p. m. - 5 p. m. Surgical Pathological Conference. Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

Thursday, March 18—

*8:30-9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and the Children's Hospitals, held this week at the Peter Bent Brigham Hospital.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Social Service Case Presentation. Mrs. Helen B. Hooker, Miss Elizabeth Grundy.

*3:30 p. m. Medical Clinic at the Peter Bent Brigham Hospital. Dr. Reginald Fitz.

Friday, March 19—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. The Extraordinary Mildness of Syphilis in Central America. Dr. George Cheever Shattuck.

12 m. Massachusetts General Hospital. Clinical Meeting of the Staff of the Children's Medical Service. Ether Dome.

8 p. m. Boston Pathological Society, Amphitheater, Vila Street entrance of the Children's Hospital, Boston.

Saturday, March 20—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

Sunday, March 21—

4 p. m. Free public lecture at the Harvard Medical School, Building D, Longwood Avenue, Boston. "Glands of Internal Secretion and Human Activity." Dr. Joseph C. Aub.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

March 11—Massachusetts General Hospital. Clinical Meeting of the Staff at 8:15 p. m. in the Moseley Memorial Building.

March 11—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

March 12—Boston Society for the Advancement of Gastroenterology, 12 noon, at the Carney Hospital, Boston.

March 15—Boston Medical History Club. See page 447.

March 16—South End Medical Club, at 12 noon, at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

March 18—Medical Clinic at the Peter Bent Brigham Hospital. See page 447.

March 19—Boston Pathological Society. See page 447.

March 23—Harvard Medical Society. See page 447.

March 29-31—First International Conference on Fever Therapy. See page 322, issue of February 18.

April 12-16—The Postgraduate Institute of the Philadelphia County Medical Society. See page 185, issue of January 28.

April 16—William Harvey Society, 8 p. m. in the Auditorium of the Beth Israel Hospital, Boston.

April 21-24—American Society for Experimental Pathology. See page 1075, issue of May 21.

June 8 and 9—American Board of Obstetrics and Gynecology. See page 78, issue of January 14.

July 6-11—Fifth International Congress of Hospitals. See page 37, issue of January 7.

October 5-8—American Public Health Association Meeting. New York City.

October 25-29—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

April 7—Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Francis B. Trudeau of Saranac Lake, New York. Subject: Personal Reminiscences of a Son of a Distinguished Father. (Illustrated.)

May 12—Annual Meeting. Salem Country Club. Speaker: Dr. P. P. Johnson of Beverly. Subject: A Trip to the Bahamas and Guatemala. (Illustrated.)

R. E. STONE, M.D., Secretary.
74 Lothrop Street, Beverly.

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesday of May.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 16—Danvers State Hospital, Danvers.

May 11—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

March 30—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May—Annual Meeting. Details to be announced.

Note: The Censors will meet for the examination of candidates on the first Thursday of May. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.
1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March 18—11 a. m. Brockton Hospital.
April 15—Annual Meeting. 11 a. m. Ducy Hospital.
May 20—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 31—See page 447.

April 28—Annual Meeting. Boston Medical Library. 8:15 p. m. "Problems in Surgical Diagnosis." Dr. Howard M. Clute.

CONRAD WESSELHOEFT, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

April 14—Worcester Hahnemann Hospital, Worcester. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOK REVIEWS

Studies from the Rockefeller Institute for Medical Research. Reprints. Volume 99, 1936. 583 pp. New York: The Rockefeller Institute for Medical Research.

In this volume are represented studies in Pathology and Bacteriology, Chemistry, Chemical Pharmacology, Experimental Surgery, General Physiology, Physical Chemistry, Biophysics, Animal Pathology, Plant Pathology, and the clinical studies of the hospital, chiefly dealing with Influenza Virus.

International Clinics. Edited by Louis Hamman. Volume III. Forty-Sixth Series, 1936. 339 pp. Philadelphia, Montreal, London: J. B. Lippincott Company.

This issue is characterized by articles of a varied nature. In the section on surgery are discussed malignant melanoma, subdeltoid bursitis, and a few others.

The major portion is devoted to subjects of a "medical" nature. There is an interesting study in the differentiation of Hodgkin's disease, lymphosarcoma, and aleukemic lymphadenosis. Tuberculosis of the Skin in the Negro, Undulant Fever, and Spontaneous Hypoglycemia are a few other representative titles. The authors are all well known. Unlike many of the preceding volumes, this one presents little of direct interest to the general practitioner. The internist and surgeon will, however, find this volume of distinct merit.

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